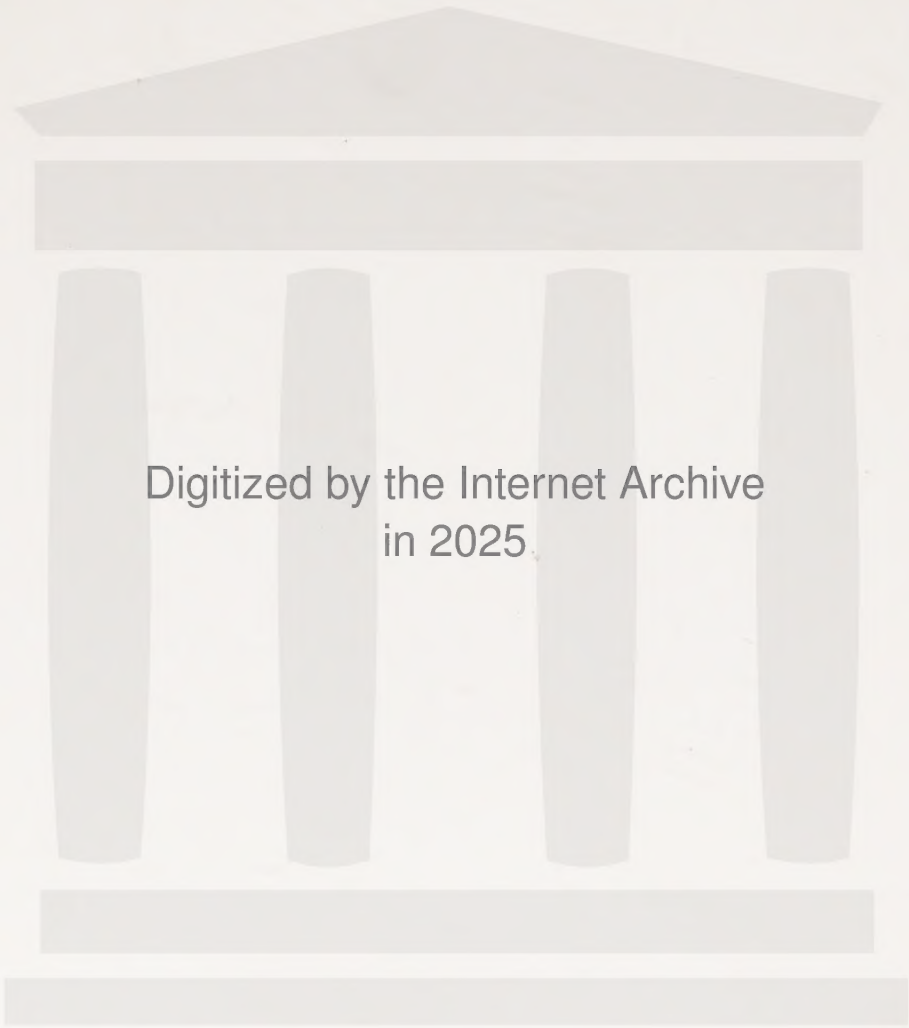
HC 1A28 7


Digitized by the Internet Archive
in 2025

https://archive.org/details/diseasesofchildr0003drmp

THE DISEASES OF CHILDREN

A WORK FOR THE PRACTISING PHYSICIAN

EDITED BY

DR. M. PFAUNDLER,
Professor of Children's Diseases, and Director of the Children's Clinic at the University of Munich.

DR. A. SCHLOSSMANN,
Professor of Children's Diseases and Director of the Children's Clinic at the Medical Academy in Dusseldorf.

ENGLISH TRANSLATION

EDITED BY

HENRY L. K. SHAW, M.D.,
Albany, N. Y., Clinical Professor Diseases of Children, Albany Medical College; Physician-in-Charge St. Margaret's House for Infants, Albany.

LINNÆUS LA FÉTRA, M.D.,
New York, N.Y., Instructor of Diseases of Children, Columbia University; Chief of Department of Diseases of Children, Vanderbilt Clinic; Ass't Attending Physician to the Babies' Hospital.

WITH AN INTRODUCTION BY
L. EMMETT HOLT, M.D.,
New York, N. Y., Professor of Pediatrics, Columbia University

IN FOUR VOLUMES

Illustrated in black and white and in colors by 61 full-page plates and 430 text cuts.

VOL. III.

BOSTON MEDICAL LIBRARY IN THE FRANCIS A. COUNTWAY LIBRARY OF MEDICINE

PHILADELPHIA & LONDON
J. B. LIPPINCOTT COMPANY

Copyright, 1908
By J. B. Lippincott Company

Electrotyped and Printed by J. B. Lippincott Company
The Washington Square Press, Philadelphia, U. S. A.

VOL. III

SPECIAL PART

(Continued)

Table of Contents

VOLUME III.

PAGE

Diseases of the Mouth 1
Dr. E. Moro, Gratz; translated by Dr. John Zahorsky, St. Louis, Mo.

Diseases of the Tonsils, Pharynx, and Œsophagus 41
Dr. H. Finkelstein, Berlin; translated by Dr. Clement F. Theisen, Albany, N. Y.

The Diseases of Nutrition in Infancy 60
Professor R. Fischl, Prague; translated by Dr. Charles Hunter Dunn, Boston, Mass.

Local Diseases of the Stomach and Intestines in Earliest Childhood 150
Professor R. Fischl, Prague; translated by Dr. J. H. Mason Knox, Jr., Baltimore, Md.

Gastro-Intestinal Diseases of Older Children 178
Professor R. Fischl, Prague; translated by Dr. J. H. Mason Knox, Jr., Baltimore, Md.

Pyloric Stenosis in Infancy 199
Professor M. Pfaundler, Munich; translated by Dr. E. J. Wynkoop, Syracuse, N. Y.

Diseases of the Appendix 213
Dr. P. Selter, Solingen, Germany; translated by Dr. James N. VanderVeer, Albany, N.Y.

Herniæ in Children 225
Dr. P. Selter, Solingen, Germany; translated by Dr. James N. VanderVeer, Albany, N.Y.

Animal Parasites 230
Dr. J. Langer, Prague; translated by Dr. Charles F. Judson, Philadelphia, Pa.

Diseases of the Peritoneum 251
Professor M. Stoos, Berne; translated by Dr. Charles F. Judson, Philadelphia, Pa.

Diseases of the Liver 267
Professor M. Stooss, Berne; translated by Dr. Charles F. Judson, Philadelphia, Pa.

The Pathology of Metabolism 278
Dr. W. Freund, Breslau; translated by Dr. Charles F. Judson, Philadelphia, Pa.

Intestinal Bacteria 288
Dr. E. Moro, Gratz; translated by Dr. Charles F. Judson, Philadelphia, Pa.

Poisons 300
Professor A. Schlossmann, Dusseldorf; translated by Dr. Charles F. Judson, Philadelphia, Pa.

Diseases of the Nose, Trachea, Bronchi, Lungs, and Pleura 311
Dr. E. Feer, Basle; translated by Dr. Theodore D. Elterich, Pittsburgh, Pa.

Diseases of the Larynx 413
Dr. Demetrio Galatti, Vienna; translated by Dr. Clement F. Theisen, Albany, N. Y.

Diseases of the Circulatory System 435
Dr. C. Hochsinger, Vienna; translated by Dr. Maurice Ostheimer, Philadelphia, Pa.

Diseases of the Thyroid Gland 524
Professor F. Siegert, Cologne; translated by Dr. S. W. Kelley, Cleveland, O.

Index 547

List of Illustrations

VOLUME III.

PAGE

1. Section Through Infant's Skull. . . . 2
2. Face of four-weeks-old Embryo. . . . 5
3. Congenital Facial Clefts. . . . 5
4. Fissure of the Lips and Palate. . . . 6
5. Maculofibrinous Stomatitis. . . . 9
6. Ulcerative Stomatitis. . . . 14
7. Thrush-Smear from the Mouth. . . . 19
8. A, B, C, Ulcera Pterygoidea. . . . 26
9. Perleche. . . . 29
10. Hutchinson's Teeth. . . . 37
11. Circular Caries of Teeth. . . . 37
12. Temperature Chart of Angina. . . . 43
13. Facial Expression in Adenoids. . . . 50
14. Exophthalmos with Adenoids. . . . 51
15. Exophthalmos with Adenoids. . . . 52
16. Pointed Cranium with Adenoids. . . . 53
17. Stricture of the Œsophagus. . . . 57
18. Advanced Atrophy in a Child. . . . 136
19. Advanced Atrophy in an Infant. . . . 137
20. Congenital Dilatation of the Colon. . . . 157
21. Congenital Dilatation of the Colon. . . . 158
22. Specimen of Dilated Colon. . . . 159
23. Congenital Atresia of the Ileum. . . . 162
24. Invagination of Cecum. . . . 168
25. Atresia of the Rectum. . . . 176
26. Peristalsis in Pyloric Stenosis. . . . 202
27. Stomach from Infant with Pyloric Stenosis. . . . 205
28. Stomach in Systolic Contraction. . . . 205
29. Chart Showing Mortality After Operations in Pyloric Stenosis. . . . 211
30. Extension of a Perityphilitic Abscess. . . . 218
31. Chart of Mild Appendicitis and Periappendicitis. . . . 220
32. Chart of Severe Perityphlitis. . . . 221
33. Congenital Bilateral Abdominal Hernia. . . . 227
34. Amœba Coli. . . . 232
35. Cercomonas Intestinalis. . . . 232
36. Trichomonas Intestinalis. . . . 232
37. Megastomum Entericum. . . . 232
38. Balantidium Coli. . . . 232
39. Eggs of Ascaris Lumbricoides. . . . 234
40. Oxyuris Vermicularis. . . . 236
41. Eggs of Oxyuris Vermicularis. . . . 236
42. Eggs of Trichocephalus Dispar. . . . 238
43. Anchylostomum Duodenale. . . . 238
44. Eggs of Anchylostomum Duodenale. . . . 238
45. Head of Tenia Solium. . . . 241

PAGE

46. Single Segment of Tænia Solium ... 241
47. Eggs of Tænia Solium ... 242
48. Head of Tænia Saginata ... 242
49. Single Link of Tænia Saginata ... 242
50. Head of Bothriocephalus Latus ... 243
51. Links of Bothriocephalus Latus ... 243
52. Egg of Bothriocephalus Latus ... 243
53. Head of Tænia Cucumerina ... 244
54. Link of Tænia Cucumerina ... 244
55. Eggs of Tænia Cucumerina ... 244
56. Tænia Nana ... 245
57. Tænia Echinococcus ... 247
58. Echinococcus Compositus Hydatidosus ... 248
59. Echinococcus Ectogenus ... 248
60. Section Through Echinococcus Multilocularis ... 249
61. Echinococcus Hooklets ... 249
62. Encapsulated Streptococci—Coli Peritonitis ... 256
63. Chronic Tuberculous Ascites ... 262
64. Caseous Peritonitis ... 264
65. Cirrhosis of Liver ... 273
66. Cystic Liver ... 276
67. Transverse Section of the Thorax ... 314
68. Temperature Chart in Double Bronchopneumonia ... 345
69. Bronchitis Croup Kettle ... 348
70. Oxygen Inhalation ... 352
71. Diagram of Frequency of Croupous Pneumonia ... 363
72. Herpes Labialis in Croupous Pneumonia ... 365
73. Temperature Chart of Croupous Pneumonia ... 371
74. Temperature Chart of Croupous Pneumonia ... 372
75. Temperature Chart of Croupous Pneumonia ... 373
76. Temperature Chart of Croupous Pneumonia ... 374
77. Chart Showing Pleurisy with Slight Effusion ... 394
78. Chart Showing Pleurisy with Large Effusion ... 395
79. Exploratory Puncture of Chest ... 403
80. Aspirator ... 406
81. Heber Drainage in Empyema ... 408
82. Rib Shears ... 409
83. Sagittal Section of Infant's Larynx ... 414
84. Sagittal Section of Adult Larynx ... 414
85. Preparation Showing Position of Thymus Gland ... 427
86. Chart Showing Average Size of Thymus Gland ... 428
87. Diagram of the Fœtal Circulation ... 438
88. Diagrams Showing Radiographic Relations of Normal Chest Shadow in Infants ... 450
89. Radiograph of Chest of Normal Three Weeks Old Infant ... 451
90. Radiograph of Chest of Normal Child Ten Years Old ... 452
91. Diagram Showing Thorax of Infant with Hyperplasia of the Thymus and Hypertrophy of Heart ... 458
92. Clubbing of Fingers in Congenital Heart Disease ... 464
93. Radiograph of Infant with Hypertrophy of Both Sides of Heart ... 469
94. Radiograph of Child with Congenital Pulmonary Stenosis ... 471
95. Radiograph of Child with Hypertrophy of Both Sides of Heart ... 472
96. Diagram Showing Isthmus Stenosis of the Aorta ... 476
97. Diagram Showing Aortic Stenosis with Open Ductus Botalli ... 476
98. Radiograph Showing Cor Bovinum with Transposition of Vessels ... 477
99. Congenital Transposition of Large Vessels ... 479
100. Opened Left Ventricle of Same Case ... 479

PAGE
101. Opened Right Ventricle of Same Case ... 480
102. Diagram of Simple Patulous Ductus Botalli ... 481
103. Patulous Ductus with Rudimentary Aorta ... 481
104. Patulous Ductus with Transposition of Large Vessels ... 481
105. Radiograph of Patulous Ductus Botalli ... 482
106. Diagram Showing Dilatation of Ductus Botalli ... 484
107. Radiograph Showing Dilatation of the Pulmonary Artery ... 485
108. Radiograph of Displaced Cor Bovinum ... 495
109. Case of Uncompensated Mitral Insufficiency ... 508
110. Congenital Goitre ... 528
111. Cretin From Thuringen ... 529
112. Congenital Myxidiocy ... 535
113. Case of Myxidiocy Before Thyroid Treatment ... 536
114. Case of Myxidiocy After Thyroid Treatment ... 536
115. Case of Myxidiocy After Thyroid Treatment ... 537
116. Case of Myxidiocy After Thyroid Treatment ... 537
117. Case of Myxidiocy After Thyroid Treatment ... 539
118. Infantilism ... 540
119. Mongoloid ... 543
120. Radiograph of Hand of Mongoloid ... 544
121. Radiograph of Hand of Cretin ... 544
122. Micromelia ... 545
123. Micromelia ... 545

LIST OF PLATES

34. (*a*) Subglossitis with Diphtheria-like Membrane; (*b*) Stomatitis Ulcerosa; (*c*) Thrush ... 13
35. Noma of the Face ... 17
36. Thrush Fungus in the Tissues ... 23
37. Affections of the Tonsils ... 45
38. Stools in Breast-fed Infants ... 95
39. Specimens from Colitis ... 187
40. Histologic Changes in the Large Intestine ... 191
41–42. Pathology of the Gastro-enteric Tract ... 195
43. Congenital Dilatation of Colon ... 156
44. Malformations of Intestine ... 175
45. Diseases of the Appendix ... 215
46. Intestinal Bacteria ... 289
47. Section Through Pneumonic Lung ... 341
48. Specimens of Pneumonic Lungs ... 363

The Diseases of Children

DISEASES OF THE DIGESTIVE SYSTEM

DISEASES OF THE MOUTH

BY
Dr. E. MORO, of Gratz

TRANSLATED BY
Dr. JOHN ZAHORSKY, St. Louis, Mo.

I. PHYSIOLOGICO-ANATOMICAL INTRODUCTION

While the structure and the functions of the mouth in later childhood approximate the conditions of the adult, the oral cavity in young infants possesses some special peculiarities. The cavity of the mouth is very small and narrow, conditioned chiefly by the lack of teeth, so peculiar to this early age, and the strong development of the tongue.

The *salivary secretion* is very scant during the first weeks of life, in consequence of which the mouth is relatively dry. With the development of the first set of teeth the secretion of the saliva becomes more active, because the upward growth of the teeth produces a sensible irritation of the dental and alveolar branches of the trigeminus and reflexly stimulates the salivary glands to activity. In the second half-year a marked increase in the quantity of the saliva accompanies the eruption of the teeth.

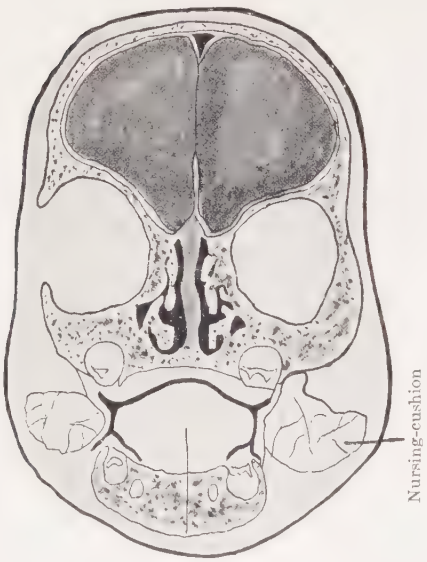
The *ptyalin* of the saliva is demonstrable even in the newborn infant (Schilling), but does not exhibit any great diastatic power until near the end of the second month (Zweifel, Korowin).

In general the reaction of the oral fluid is appreciably acid, while the product of the salivary glands obtained with strict precautions gives a weak alkaline reaction. The acid reaction is produced by the fermentative processes of bacteria, which find favorable conditions for life in the warmth of the buccal cavity and in the presence of milk particles.

The characteristic ingredient of the saliva in the adult, namely, potassium sulphocyanide, is entirely absent in the secretions of the mouth in the first months of life.

The absence of teeth and the deficient salivary secretion plainly show us that the organs of the mouth in the infant can not meet the demands which are required at a more mature age. On the other hand, we recognize precisely in this backward development a judicious arrangement for the *act of nursing*, the most important function of the infant's mouth. The several organs of the mouth in the normal infant are more or less specifically modified in this direction. Not only the fleshy, bulky character of the tongue, which makes the inspection of the faucial walls difficult, but also the strong resistance which the masseters of the newborn infant usually show on attempting to open the mouth forcibly, indicate that the organs concerned in the act of sucking are already highly developed at this very early period.

Fig. 1.



Section through the skull of an infant two months old, one-half natural size.

Ranke has endeavored to explain the so-called "*corpusculum adiposum*" as a supporting organ to the act of sucking. This fatty body in the cheek of infants was first described by Geheve, a pupil of Walter. It is a circumscribed mass of adipose tissue, supplied with a movable attachment, and on both sides it is so situated that it lies immediately over the buccinator and masseter muscles. According to Ranke, its biological significance is that of a nursing-cushion ("saugpolster"); in other words, it serves as a bolster for the buccinator; by stiffening its substance it gives support to the function of the cheek during the act of nursing (Fig. 1).

Indeed, it is very remarkable that this accumulation of adipose tissue, which is especially noticeable in atrophic infants, should retain its original volume even in cases of extreme emaciation; which fact, judging from the gradual involvement of the organs in the process of inanition, seems to indicate that it has some function very essential to life. This is the chief reason why Ranke assumed the nursing-cushion to have a very important function which otherwise would seem obscure judging from the modern view of the nursing mechanism.

Furthermore, a judicious adaptation to the act of sucking is found in the strong development of the levator labii superioris muscle and the ragged prominences on the inner mucous membrane of the lip which are about 1 mm. in length and were first described by W. Krause. These atrophy after serving the purpose of applying the mouth closely to the nipple.

The *mechanism of the sucking act* (Auerbach, Escherich, Pfaundler) occurs in two alternating stages. The first stage consists in the depression of the tongue and lower jaw, after the jaws, tongue and lips have enclosed the nipple airtight. Through this act the oral cavity is enlarged and, as the dependent soft palate closes the faucial opening, a negative pressure is produced in the mouth. The first action serves to grasp the mammilla and draws the milk into the outer lactiferous passages.

The second stage consists in the closure of the jaws. This compresses the nipple, which lies embedded in a groove of the tongue, and forces the milk into the mouth. The negative pressure induced by the act of sucking has been measured by Pfaundler. It is possible that a sum of successive sucks are produced, whereby the pressure may rise to 30 cm. (water or milk in the manometer); in older and vigorous infants it may attain even a pressure of 70 cm.

The act of sucking is the only important function which may be assigned to the mouth organs of the nursling. All other mechanical functions, such as the amylolytic activity of the saliva and the detergent properties of the secretions, are superfluous for the infant nourished exclusively on milk. It is conclusive, therefore, that the mouth of the infant, as a special organ of digestion, holds a subordinate position, but rather assumes the rôle of an indifferent passage-way for the milk.

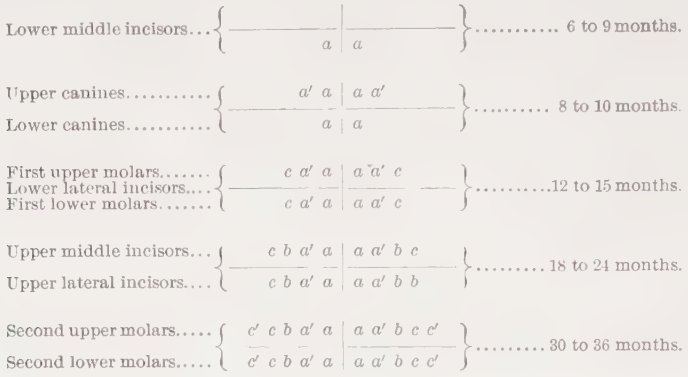
The inability to perform the act of sucking may obviously endanger the life of the infant. As diseases of the mouth are very frequent antecedants of the inability to nurse, the importance of a knowledge of its diseases and their treatment is clear; and, for reasons mentioned, even insignificant lesions require the attention of the physician.

The *mucous membrane* of the infant's mouth is tender, easily vulnerable, and, consequently, the frequent seat of superficial lesions. In spite of a strong healing tendency, these minute defects serve, under certain conditions, as a nidus for a local bacterial invasion which by extension occasionally produces serious grades of disease and may even lead to sepsis. The most frequent source of injury to the mucous membrane is found in the manipulations performed in cleansing the mouth.

The so-called *epithelial pearls* are very singular anatomical structures of the gums. On account of the frequency of their presence (over 90 per cent.) they must be regarded as a physiological appearance, although they must be considered as minute retention-cysts of the mucous glands. They are filled with pavement epithelium. These anatomical peculiarities are often called Bohn's nodules after the author who first described them. They are commonly situated in or beside the raphe, also in the upper posterior alveolar border and on the hard palate. They vary in size from a millet seed to a pinhead and present a shining milky or yellowish color. The nodules gradually disappear by spontaneous absorption or by ulceration and discharge of their contents.

NORMAL DENTITION

The following table shows the physiological limits in the eruption of the milk teeth:


Lower middle incisors... a | a ..... 6 to 9 months.
Upper canines..... a' a | a a'
Lower canines..... a | a ..... 8 to 10 months.
First upper molars..... c a' a | a a' c
Lower lateral incisors.... c a' a | a a' c .....12 to 15 months.
First lower molars.....
Upper middle incisors... c b a' a | a a' b c
Upper lateral incisors.... c b a' a | a a' b b ..... 18 to 24 months.
Second upper molars..... c' c b a' a | a a' b c c'
Second lower molars..... c' c b a' a | a a' b c c' ..... 30 to 36 months.

The *second dentition* begins in the fifth or sixth year with the eruption of the third molars. The deciduous teeth drop out approximately in the same order in which they appeared. In the twelfth year the fourth molars and, lastly, in the sixteenth to twenty-fourth year the fifth molars, the so-called wisdom teeth, pierce the gums.

THE CARE OF THE MOUTH

The care of the mouth in healthy persons has the same significance as the *care of the teeth.* From this principle, which has been formulated by the profession, the following conclusion may be inferred: The toothless mouth of the infant needs no special care.

Earlier than this it has been asserted that the lesions and infections of the mucous membrane of the mouth were the immediate results of the manipulations used in cleansing the mouth. For this reason the washing and wiping of the oral cavity must be rejected as an unnecessary and harmful procedure (Epstein). The practical application of this rule is everywhere followed by the best results and can scarcely be emphasized too much; the cases of mouth diseases in infancy are very much reduced in number when less attention is given to the mouth.

In the case of the child beyond the age of infancy the hygienic rules useful for the civilized adult are indicated. The regular care must commence with the milk teeth, because, as experience has taught, the health of the permanent teeth depends in a great measure on that of the deciduous teeth. Furthermore, the milk tooth gives the permanent tooth its direction of growth; wherefore, the former should be retained as long as possible.

The greatest danger to the teeth arises from the food particles which cling to their surface, and which by fermentation gradually dissolve

the inorganic substance of the tooth; the bacteria of decay only then begin a successful work of destruction. The chief hygienic rule, then, is to remove all the food particles after each meal. This demand can usually be fulfilled by the use of the toothpick, by cleansing the crown with the tooth brush, and by flushing the mouth with water.

The proper use of the toothbrush should be taught to the children as early as possible. The toothbrush should be soft, and when brushing the teeth should be moved in a vertical direction parallel to the interdental spaces. Brushing the teeth transversely does more harm than good. The subsequent washing of the mouth should be done with a sucking movement in order that the water may be forcibly whirled between the teeth.

The cheapest and most rational cleansing preparations are prepared chalk and lukewarm water. The usefulness of antiseptics is doubtful; the detrimental effect of tooth soap is certain.

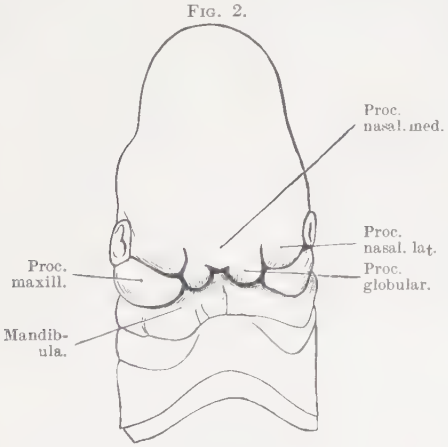
Even in periods when no disturbances referable to the teeth exist, the child should be sent to the dentist for inspection at least once a year.

The care of the mouth demands special attention during the course of diseases, particularly those of an infectious nature, which rule can scarcely be made too important.

II. CONGENITAL MALFORMATIONS

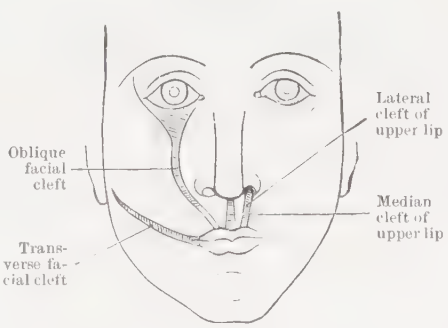
The oral cavity, or to be more exact, the bones and integument having a part in the structures of the mouth, are frequently the seat of congenital malformations in the form of abnormal clefts.

Fig. 2.



Face of an embryo about four weeks old, schematically represented.

Fig. 3.



Scheme of the congenital facial clefts.

In order to understand the origin of these fissures the adjoining illustrations will be found helpful. Fig. 2 represents the face of an embryo about 4 weeks old. Fig. 3 is a diagrammatic representation of the most

common congenital malformations. A comparison of the two figures will show the origin of some congenital fissures. It is obvious that the mesial fissure of the upper lip is produced by a failure in the fusion of the globular processes. The transverse cleft is a consequence of failure in the obliteration of the fissure between the maxillary and the mandibular processes. The lateral fissure of the upper lip, or harelip, indicates that the cleft between the globular and maxillary processes has failed to close; that is, the single lower and small inner limb of the Y-shaped cleft system illustrated in Fig. 2. The oblique facial fissure arises from the cleft which extends between lateral nasal process and the maxillary process in the direction of the orbit; that is, the lower single and the external larger limb of the Y-shaped cleft system.

Fig. 4.

Fissure of the lips and palate.

Harelip (*labium leporinum*) in its various grades is the most frequent of the congenital malformations. The simplest grade occurs as a depression in the upper lip near the philtrum, which in the form of a glistening streak occasionally extends to the nostril (partial harelip). As a rule the fissure extends to the nostril (partial harelip, second degree), or is prolonged into the nose, so that its upper end is not visible (total harelip). Often the harelip occurs on both sides and very often it is combined with a malformation of the maxilla.

The prognosis of harelip depends primarily on the extent of the malformation and the strength of the affected infant. As the alimentation is often hindered and the exposed mucous membrane is subjected to inflammatory processes of various kinds, the question of operative intervention must be considered very early (second to fourth week). A more unfavorable prognosis is submitted by those faucial clefts which are combined with malformation of the palate. The most common combination is that known under the name of wolf's throat (see Fig. 4).

The *wolf's throat* consists in a cleft of the hard palate, which is divided by the vomer into two fissures. This cleft may extend posteriorly in the median line through the soft palate and anteriorly terminate in a double harelip, between which the free intermaxillary bone pro-

trudes. This malformation represents the extreme grade of cleft palate. Defects or fissures of the palate are frequently observed and give the voice a peculiar nasal twang. These must be attributed to a palatal cleft which has partially healed during intra-uterine life.

The serious derangements which result from congenital defects of the palate embrace, above all, the functional disorder of the act of nursing and deglutition. Although the milk may be placed on the dorsum of the tongue by means of a spoon or dropper to stimulate the act of swallowing, a large portion of the food, fed with difficulty, flows outward again, and it is necessary to resort to gavage in order to save the infant from starvation.

The danger of an inflammatory process attacking the mucous membrane is greater in these cases than in harelip, since the nose and mouth communicate freely. Even before an operation can be undertaken, these unfortunate infants usually succumb to an intercurrent affection, such as a bronchopneumonia, or a digestive disorder induced by swallowing food particles which have undergone putrefaction in the nose.

The cause of this malformation must be sought in some hindrance to proper development, mostly some mechanical cause; as an abnormal embryonic displacement of the base of the skull, an abnormally developed amnion or an increased intra-cranial pressure. A primary atrophy of the borders, a possible result of an inflammatory process which prevents fusion, has also been designated as a possible cause. Heredity plays a most important rôle in these as in all congenital deformities.

III. PRIMARY AFFECTIONS OF THE MOUTH

Under the name primary affections of the mouth are grouped those inflammatory processes which arise independently of any other disease of the infantile organism.

This group embraces a series of inflammatory processes, clinically and anatomically well defined which are classified under the term "*Stomatitis.*" The classification of the stomatitides, with the exception of thrush, must be based entirely on clinical and anatomical characters; since the etiology in some of the diseases is entirely obscure, in others still insufficiently studied, and consequently uncertain.

We therefore distinguish catarrhal stomatitis, aphthous stomatitis (maculofibrinous stomatitis), herpetic stomatitis, ulcerative stomatitis, and gangrenous stomatitis (noma). To this list must be added the stomatitis due to thrush fungus and ulcera pterygoidea (Bednar's aphthæ).

As may be inferred from the foregoing, the clinical varieties of stomatitis may assume different degrees of intensity. We know clinical forms of inflammation which appear in the forms of a catarrhal irritation and quickly heal spontaneously; also the severe destructive stomatitis which under the syndrome of a genuine gangrene rapidly ends in death.

CATARRHAL STOMATITIS

Nature, Local Changes.—Catarrhal stomatitis is characterized clinically by the special symptoms of inflammation, namely, redness, swelling, pain and a hypersecretion of the affected mucous membranes. A dark red color, as a rule, precedes an evanescent grayish white discoloration produced by cloudy swelling of the superficial epithelial layer. After desquamation of the epithelium an intense red color remains throughout the course of this benign disease. As a rule, the disease involves chiefly the gums and the tongue. Occasionally the lips, the cheeks and the palate are also implicated, so that the lining of the whole oral cavity is more or less intensely inflamed. The inflammation is usually limited to the oral cavity without spreading to the structures of the pharynx.

Symptoms.—Attention to the mouth is most commonly directed by the fact that children have pain during eating, and for this reason it is at times necessary to stop feeding. In recognition of this fact it is best to give the children only cool and non-irritating fluids.

The salivation, which accompanies the inflammation, is sometimes so profuse that the saliva flows from the corners of the mouth and irritates the external surface. A sensitive skin reacts with a more or less intense redness. The temperature, as a rule, is normal or very slightly elevated. Swelling of the lymphatic nodes is absent. Nevertheless, the general condition suffers, as is evidenced by the stationary weight, or even loss, and the evident lassitude and irritability of temper.

Etiology.—The catarrhal inflammation of the mouth arises mostly as a result of local irritants of a mechanical, chemical, or thermic nature. This disorder precedes and attends all other forms of stomatitis.

Among the mechanical causes a prominent part is played by the irritation that attends the eruption of the milk teeth; for it cannot be considered entirely accidental that stomatitis most frequently occurs during the period of dentition (first and second year).

Therapy.—No special treatment is required, since spontaneous recovery occurs in a few days.

MACULOFIBRINOUS STOMATITIS (APHTHOUS STOMATITIS)

In the following description the old term aphthous stomatitis is avoided and a new name is introduced. This is done with the best intentions. Without considering the fact that the term aphthæ has wrought irremediable confusion, it explains nothing; for the translation of stomatitis aphthosa is "inflammatory inflammation of the mouth," as ἄφθαι is derived from ἅπτειν, that is, to inflame.

In this usage I follow the example of E. Fränkel and Kraus, who proposed the term "stomatitis fibrinosa maculosa disseminata," and

in order to shorten the term, I shall employ the name maculofibrinous stomatitis, which has the same significance.

The old physicians applied the word aphthæ to all diseases which were characterized by the formation of a white deposit on the mucous membrane with or without loss of substance. It is obvious that this broad view embraced a large number of different diseases. So it happened that genuine diphtheria of the mouth or throat, as well as all necrotic and ulcerative processes of the mouth, even thrush, were designated aphthæ. Only after recognition of the various causative factors, did the clinical conception of aphtha become limited to a lesser field, and finally, without any special reason was reserved for a certain inflammation of the mouth characterized by the formation of disseminated yellowish white plaques in the superficial layers of the mucosa.

Nature, Pathogenesis.—The appellation maculofibrinous stomatitis clearly expresses the nature of the disease. We have to deal with a deposition of a fibrinous exudate in the superficial layers of the mucous membrane, as Henoch has already taught. The eruptive lesions, which appear as small round spots, are well defined and have a whitish color. Their periphery shows a reddish areola. The patches at first lie subepithelially upon the inflamed layers of mucous membrane. Soon the epithelial cover bursts and the yellowish white, disintegrated mass lies free upon the mucous membrane, occasionally surrounded by loosened epithelial cells (Fig. 5).

Fig. 5.

Maculofibrinous stomatitis.

The histological investigation of the lesions permits the conclusion that the morbid changes must be identified anatomically with the fibrinous pseudomembranes, as they occur typically in croup and diphtheria. Degenerative processes in the sense of a necrosis are entirely absent, for, without considering the fact that such tissue alterations are not observed, after the absorption of the exudate no ulceration remains, except a superficial erosion which heals without a scar.

The reparative process occurs in that the floor of the deposit cleans off, the disintegrated masses are absorbed, and at the same time new

epithelium is formed at the periphery for the purpose of covering the erosion. It must be emphasized that the described efflorescences of maculofibrinous stomatitis arise directly and are never preceded by an eruption of vesicles. Herpetic stomatopharyngitis, and foot-and-mouth disease must be carefully differentiated from maculofibrinous stomatitis, although the former diseases sometimes, when the vesicles rupture and the white base is exposed, may resemble the latter affection.

Symptoms, Localization.—Maculofibrinous stomatitis is characterized by the appearance of discrete, yellowish white spots on an inflamed mucous membrane. The spots vary in size, from a hemp-seed to a pea, and may occur on the lips, the cheeks, or the palate. Not infrequently two adjoining spots coalesce whereby longer and irregular patches are formed. The plaques, of course, adhere firmly to their base; if they are forcibly detached their bed bleeds very slightly.

The *symptoms* are those which we have already learned in discussing simple stomatitis, but in greater severity. Usually severe burning pains are felt in the mouth and the children take food with great discomfort. There is an increased flow of saliva which does not possess a disagreeable odor. The general condition also suffers in a great degree. The children are weak, irritable and have no appetite. Their sleep is restless and disturbed. Occasionally diarrhœa supervenes. Whether this is due to the ingestion of large quantities of saliva may be considered doubtful. Not infrequently the maxillary lymph-nodes are found enlarged and, at the onset of the disease, a febrile movement may be discovered.

The course of the disease is always favorable. As soon as the local inflammatory changes cease, the general condition improves. The febrile movement ceases, the appetite and proper sleep return, and after a week, if no complications arise, recovery supervenes to proper treatment.

Etiology.—The cause of maculofibrinous stomatitis is uncleanliness of the oral cavity, inadequate care of the mouth, the use of filthy "soothers" and unclean utensils. The disease, therefore, occurs chiefly among the children of the proletariate and is rarely observed among the higher classes. The fact that most cases occur during the second year of life suggests some relationship to teething. Indeed, in some cases the disease is observed to arise at the time of the eruption of a tooth, as a complication of the catarrhal stomatitis. Fibrinous deposits appear and wreath-like encircle the crown of the new tooth. Otherwise, the fact that infants, when they commence to walk, crawl on all fours, must be considered since the hands are soiled and may directly infect the mouth (Escherich's dirt infection).

These factors argue that maculofibrinous stomatitis has an infectious origin, especially since the sudden appearance of the eruption is attended by fever and, moreover, many cases have been reported which establish the communicability of the disease.

Investigations concerning the nature of the exciting cause have, as yet, not yielded any definite results. Staphylococci have been found most frequently, but the etiological relation has not been established since they form a part of the normal flora of the oral cavity. Stoos was able to demonstrate in a number of typical cases the presence of a large diplostreptococcus, which very much resembled Tavel's *diplococcus intestinalis major*. Since this diplococcus is constantly present in the disease and constantly absent from the healthy mouth and, moreover, since the microörganisms have a crowded arrangement in the exudate, Stoos insists that the diplococcus is the specific cause of maculofibrinous stomatitis. Still, the fact that a long diplostreptococcus is often found in microscopical preparations of faucial exudates and in the stools of infants warns us to be cautious in accepting this finding as final, especially since positive inoculations have not been made.

The etiological relation of "foot-and-mouth" disease has often been discussed. It is more than probable, however, that this disease, both etiologically and clinically, is quite distinct from maculofibrinous stomatitis and must be regarded as a distinct disease entity.

Diagnosis.—Maculofibrinous stomatitis is adequately characterized by its appearance and the seat of the lesions, so that the diagnosis as a rule offers no difficulties. In the differential diagnosis only herpetic stomatopharyngitis and small decubitus ulcers of genuine ulcerous stomatitis need be considered. The involvement of the throat (especially the pillars of the fauces) and the possible presence of recent vesicular lesions would point to the herpetic sore-mouth; while in the case of ulcerous stomatitis the localization of the deposits (around the teeth, gums, and edge of the tongue) and the fetor of the secretions, are diagnostic, the foul breath usually being absent in maculofibrinous stomatitis.

Like herpetic stomatopharyngitis, the foot-and-mouth disease arises by the eruption of vesicles, which may attain the size of a pea. This vesicular eruption is not limited to the oral cavity, but appears also on the integument particularly in the neighborhood of the nose and mouth. Although the foot-and-mouth disease usually assumes a milder form in children than in the adult, still the local and general symptoms (especially in regard to the alimentary canal) are much more severe than in maculofibrinous stomatitis. A common peculiarity of the two diseases is that no destruction of the deeper tissues takes place. In doubtful cases the previous history (such as ingestion of raw milk from diseased cows) must decide the question.

The **prognosis** is invariably good.

The **prophylaxis** consists in a rational care of the mouth, in the prevention of contact, and the avoidance of unclean "soothers" and filthy eating and drinking utensils.

Treatment.—For local use in older children a solution of potassium chlorate (2 per cent.) or boric acid (2 per cent.) should be prescribed. In young children the potassium chlorate should be given internally (1–2 per cent.) a teaspoonful every two hours. In obstinate cases the local application of a 2 per cent. solution of silver nitrate, carefully applied with a brush, is indicated and should be used once or twice daily.

In order to spare unnecessary pain, it is best to give cool liquids and to exclude solid and acid foods from the diet.

ULCERATIVE STOMATITIS (STOMACACE)

For a long time ulcerous stomatitis was regarded as a buccal manifestation of genuine diphtheria, as the classical writer on diphtheria, Bretonneau, himself proclaimed this erroneous conception. The views in regard to the nature of this disease went through numerous changes, but it was finally recognized as a distinct form of stomatitis (Bergeron, 1859), when the identity of an epidemic of stomatitis among the troops of Paris with the ulcerative disease of the mouth in children (which could frequently be observed in hospitals) was established.

Pathogenesis, Local Symptoms, Localization.—Ulcerative stomatitis (Plate 34) depends on the presence of teeth and almost always arises on the gums. The gums swell cushion-like and assume an inflammatory redness. The redness soon yields to a more livid discoloration of the affected part. The gum surrounding the tooth loosens its attachment and begins to separate its swollen border completely from the tooth. Following this, a yellowish discoloration appears on the edge of the gums, which is produced by a purulent exudate in the superficial layers of the mucous membrane. Beneath this exudate the tissues undergo a necrotic process and in a short time we find an ulcer in place of the infiltrated margin. The ulcer begins to grow rapidly and may extend to the buccal portion of the gum. As a consequence the larger part of the root is denuded and the tooth itself rests loosely in the alveolar socket.

The exudate has a yellowish, brownish, or dirty color and adheres firmly to the floor of the ulcer. If it is detached the base bleeds. Especially characteristic is the fetid odor of these greasy masses, which is also conveyed to the breath and to the increased secretions of the mouth. This penetrating fetid odor, occasionally perceptible at a distance, is very characteristic of the disease.

The morbid process only exceptionally remains limited to the gums; in most cases it attacks the regions of the mouth adjoining the primary foci. Consequently, we observe the neighboring mucous membrane of the lips or cheek, as well as the edge of the tongue undergoing similar alterations; and it is remarkable that the resulting sores are a real counterpart in shape of the original ulcers on the gums. We find

a. Subglossitis with diphtheria-like membrane in a 3-year-old boy with pertussis. Little local reaction. No fever. Spontaneous dropping of fibrinous exudate.

b. Stomatitis ulcerosa in an older child caused by caries of the back teeth.

c. Thrush. From a 5½-months-old atrophic child. Growth on the tongue, cheeks, gums, etc. The entire mucous membrane is inflamed.

these secondary lesions occurring especially typical on the edges of the tongue, which consequently has a marked indented aspect. In their nature, the secondary ulcers are decubitus sores, but still their specific character allows furthermore a genuine contact infection. The tongue is covered with a thick, slimy coat. At the same time a swelling of lips and cheek supervenes which may often be perceptible from the outside. The neighboring lymphatic nodes undergo an obstinate enlargement.

The region of the lower jaw is oftener affected than that of the upper jaw. The palate is generally exempt, but in certain cases deposits are found on the soft palate, especially the tonsils, which in appearance are identical with the pultaceous exudate of ulcerative stomatitis and which lead to a destruction of the tonsillar tissue (angina ulcerosa). If the mouth remains free from the disease, this form of angina may be mistaken for diphtheria (see chapter on angina).

Very interesting in this connection is a singular case reported by Bernheim and Pospischill in which an ulcerative stomatitis, without infecting the tonsil, spread to the larynx and actually produced stenosis.

Microscopical Findings.—If scrapings from an ulcer are examined microscopically the exudate is seen to be composed of detritus, pus-cells, disintegrated blood corpuscles, and epithelium, as well as numerous microörganisms. A stained preparation reveals in every case a typical bacteriological picture which will be described later.

General Symptoms.—The general condition of the child is severely affected by the disease. As early as the first stage we observe that the children are noticeably depressed, irritable, and complain of severe pain in the mouth. These symptoms increase in severity when the first ulcers are formed. Febrile movement supervenes regularly with these symptoms. The children are entirely without appetite and the ingestion of food is almost impossible on account of the local pain. From this condition, also from the general influence of the disease, the patients become very pale in the course of the disease; in fact, they often give the impression that they are suffering from some serious malady. After recognition of these facts, the opinion that an autointoxication is produced from the ingestion and resorption of the foul disintegrated masses cannot be denied.

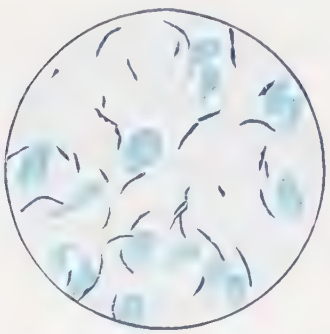
Course of the Disease.—In a normal course, the ulcerations begin to heal after 6 to 10 days. The exudate peals off, the tendency to bleed ceases, and the reparative process follows in the form of a rapid cicatrization from the periphery to the centre. With the clearing of the ulcers, an amelioration of the symptoms progresses step by step. The tenderness lessens, the taking of food is easier, and the children assume quite a different aspect.

Occurrence.—The disease is generally disseminated and by no means rare. The endemic outbreaks of the affection in closed asylums

are remarkable and have been extensively studied (Bernheim, etc.). Because the occurrence of the disease pre-requires the presence of teeth, the disease does not occur in the young nursling. Primary ulcerative stomatitis is observed most frequently about the time of the second dentition, that is, about the seventh year, and at the time of the eruption of the first molars, that is, about the twelfth or thirteenth year.

Etiology.—Caries of the teeth and neglected care of the teeth play the chief rôle in the causation of the disease. Certain cachectic conditions of the young organism and its oral mucous membrane, as are often seen after the acute infectious diseases—especially measles, but also scarlatina, diphtheria, and typhoid,—seem to be very important. It is well known what a prominent part the inflammatory affections of the mouth play in the morbid conditions following measles. Not infrequently, we observe a genuine ulcerative stomatitis as a direct complication of measles (stomatitis ulcerosa cachectica). The described conditions must, however, be regarded merely as predisposing factors.

Fig. 6.



Ulcerative stomatitis. Smear from a purulent deposit. Stained with Löffler's methylene blue, enlarged about 700 diameters. Bacilli fusiformi, spirochæte, pus cells.

The constancy of a typical bacteriological finding convincingly corroborates the truth of the former statement. In smear preparations of the exudate the predominence of two kinds of bacteria which are regularly associated, attracts attention; a spindle-shaped bacillus (bacillus fusiformis), taking up the common aniline stains with avidity, and a delicate, cork-screw or screw-shaped spirochæta, which appears less distinctly stained (Fig. 6).

While both bacteria were known to Miller as a frequent finding in carious teeth and Plaut and Stooss had the opportunity to observe this microscopical picture in infectious anginas, special credit must be bestowed on Bernheim and Pospischill, who first demonstrated their causative relationship to ulcerative stomatitis.

The bacillus fusiformis in shape, arrangement, and staining capacity (vacuole formation in the plasma) has a superficial resemblance to the diphtheria bacillus and the unskilled might make an erroneous microscopical diagnosis of this disease (Bernheim). However, its larger size and its tendency to group in diplobacillary bands, but especially its pointed ends (spindle-shaped), morphologically differentiate it from the Löffler bacillus. Not infrequently the body of the bacillus shows a semilunar curve and recalls the shape of a little boat. The fusiform bacillus as well as its constant associate, the spirochæta, shows lively active movements in a native preparation. Staining is best accomplished with fuchsin or Löffler's methylene blue. Recently the artificial growth

of the bacillus fusiformis has succeeded in a serum culture medium under strict anaërobic circumstances. The cultures diffuse a fetid odor and inoculations on animals produce a local necrotic process (Ellermann). These facts, in addition to its constant presence in the mouth affected with the specific disease, amply prove its etiological relation to ulcerative stomatitis.

Diagnosis.—The appearance is so characteristic that simple inspection of the mouth permits no doubt as to the nature of the disease; for even excepting the specific qualities of the primary and secondary ulcerations, the penetrating fetor of the mouth, which is characteristic of ulcerative stomatitis, is never absent. In the differential diagnosis, the necrotic form of mercurial stomatitis and the scorbutic affection of the mouth need only be considered. In both instances the characteristic history and onset of the disease decide the question.

The **prognosis** is generally favorable, but the possibility of complications should not be forgotten. In protracted cases, it may happen that the process involves the periosteum of the maxillary bone and leads to a partial necrosis. Abscess of the tongue, angina Ludovici, general sepsis, and especially noma are among the complications which have been observed in this disease. Fortunately, such cases belong to the greatest rarities.

Prophylaxis.—A rational care of the mouth, especially the teeth, stands foremost among the prophylactic measures. Particularly during the course and decline of the infectious diseases mentioned (measles and typhoid particularly) attention to the hygiene is very necessary. Carious teeth, the most frequent source of the evil, must be treated or extracted.

Treatment.—In the first place a vigorous local treatment should be instituted. It is necessary to prepare the ulcers for the reception of the antiseptics by a careful cleansing of the mouth. Of the medicaments to be recommended, a dark solution of potassium permanganate or hydrogen sulphide are particularly valuable. For sponging and flushing the mouth the former is to be used several times a day, the latter in 2 per cent. strength three or four times daily.

In obstinate cases, if this does not succeed, the direct application of iodoform gauze soaked with aluminum acetate may be tried. Pencilling the ulcers with zinc chloride (5 per cent., 2 times daily) acts almost as well as silver nitrate and has the advantage in that it does not attack the healthy mucous membrane.

Local treatment is effectively aided by the internal administration of potassium chlorate (2 per cent., small teaspoonful every two hours).

In protracted cases the nutrition of the child needs careful attention. As has already been stated, the general condition in many cases may suffer severely. For this reason it is necessary to give the children a

nutritious liquid diet (milk with food preparations, eggs, etc.) and special vigilance must be used to see that the patients take a sufficient quantity. If this cannot be done in the natural way, one should not hesitate to resort to forcible feeding by means of the stomach tube.

STOMATITIS GANGRÆNOSA (NOMA)

By noma of the face (see Plate 35) we understand a rapidly progressive and generally fatal gangrene of the cheek, probably produced by a specific microörganism.

From the writings of the Hollander Battus (16th century) the inference can be drawn that the disease was more frequent in ancient times than at present. To-day, noma is observed very rarely and only in isolated cases. The moist and foggy coasts of the North, as Holland particularly, the coast of Northern Germany, Denmark, and England, appear to be a more frequent habitat of the disease than the southern countries.

Pathogenesis, Local Symptoms.—Two qualities are especially characteristic of the disease: first, that children exclusively are attacked; and, second, that this process, almost without exception, occurs in a body which has been injured and exhausted by some previous disease. The diseases which may predispose to noma are the following: measles, then diphtheria, typhoid, scarlet fever, severe enteritis and dysentery, cerebrospinal meningitis, pneumonia, and tuberculosis. As has been stated, noma may result from ulcerative stomatitis in isolated cases. Genuine cases of idiopathic gangrenous stomatitis are known (Billard, Henoch) to have arisen spontaneously.

The first symptom noticeable externally is a severe swelling of the side of the face, which is marked by a fatty gloss and a waxy pallor. On palpating the affected part, which contrary to the expectation is not very painful, a hard diffuse infiltration in the depth of the tumor is found. On inspecting the oral cavity, a small ulceration, which had its origin in a bleb, and which is covered by a blighted, greenish gray mass, is seen on the inner surface of the affected cheek. Even at the beginning of the disease, a nasty fetor is exhaled from the mouth, which increases in intensity as the disease progresses. The submaxillary lymphatic nodes are swollen.

The gangrenous ulceration grows perceptibly from hour to hour, both in breadth and in depth, and soon at the point of the outer cheek corresponding to the deepest part of the ulceration, a rapidly spreading red spot appears, which becomes darker and takes on a black and blue discoloration and finally changes to a dry, fissured, blighted scab. This necrotic area may reach to the eye and downward to the neck. A pale red zone of demarcation surrounds the scab, which either becomes perforated in places, or is totally cast off. The resulting defect, from

PLATE 35.

October 12, 1906.

October 14, 1906.

October 15, 1906.

October 18, 1906.

October 22, 1906.

October 22, 1906, death.

NOMA, 7-YEAR-OLD BOY.

which putrid and bloody masses are discharged, sometimes permits direct vision into the oral cavity.

Meanwhile, as a rule, the progressive gangrene has produced great destruction in the mouth. The neighboring gum rots off, the periosteum dies; consequently, the alveolar process becomes denuded and the teeth loosened, so that they may fall out.

Localization.—It is a remarkable fact that the process is almost always limited to one half of the face. Exceptionally, cases of noma have been observed where the necrotic process began on the outer surface of the cheek. Whether these cases are genuine noma, or some symptom-complex which simulates it, can not be decided at present. Rare localizations of noma are the external ear and the vulva.

General Symptoms, Course.—Not seldom an astonishing contrast is observed between the wide-spread gangrene and the subjective symptoms of the spreading local process; the children in many cases still find pleasure in play and still have appetite, both undoubted signs that the body is in less danger than is really expected. Soon however the picture changes. High fever sets in and the ingestion of food becomes less and less. As a rule profuse diarrhœa supervenes (probably autotoxic); delirium and collapse follow and death results with the symptoms of general exhaustion; if some complication, as pneumonia or abscess of the lung or other septic process, has not already relieved the patient from its sufferings.*

Etiology.—The microparasitic nature of noma is to-day probably beyond question. The endemic occurrence in asylums and the whole course of the disease support this view. Of course, the importance of a strong individual predisposition must be accentuated here more than usual.

Furthermore, the recent discoveries of bacteriological research are very convincing, inasmuch as they agree in essential points; an anaërobic bacillus has been found by all investigators. This microörganism usually grows in spiral threads (cladothrix); it exists in large numbers, especially in the inflamed tissues of the zone of demarcation, and, consequently, is generally regarded as the exciting causative agent (Schmorl, Perthes, Hofmann and Küster, Seiffert, etc.). The cultivation of this microörganism and inoculation experiments have also been successful. This discovery has become still more significant since these bacilli, morphologically and biologically, have been shown to resemble Löffler's microörganism, which causes nomatous gangrene in domestic animals (so-called calves' diphtheria).

Histological examination of the affected parts reveals the signs of a necrotic disintegration of the tissues. Very remarkable is the heavy deposit of "noma-threads" around the muscle fibres which are thus

* The four cases of Brüning ran a very protracted course, two of which ended fatally at the end of four and six weeks respectively; while of the two others, one recovered from the morbid symptoms in three and one-half months and the other after one year.

encircled by bacteria and the nutrition is thereby impeded. Another peculiarity is found in the growth of a luxurious fungus-sod on the walls and in the lumen of the blood vessels (Brüning).

Diagnosis.—A typical case presents no difficulty in diagnosis. The genuine gangrene, without any antecedent severe inflammatory process is a singular manifestation of noma and plainly distinguishes the disease. But the onset of noma does not always take such a rapid course. Occasionally the swelling of the cheek is present for several days before gangrenous changes of the tissues ensue. Especially after ulcerative stomatitis, which is often attended by a considerable infiltration of the cheek, the onset of noma may not easily be recognized. This is really unfortunate as according to all evidence early therapeutic measures are very desirable.

The **prognosis** of noma is always grave. Very few cases recover (about 15 per cent). Severe deformities and functional derangements, which need surgical treatment, are the constant results of the disease.

The **prophylaxis** is naturally limited to a careful hygiene of the mouth during the designated infectious diseases and proper medical treatment of ulcerative stomatitis.

Treatment.—Two views in regard to the methods of treatment stand opposed to each other. Most of the authors favor an early radical operation (Perthes, Ranke, Springer) others advise an expectant treatment and recommend surgical intervention only after the process ceases to spread (Soltmann). The excellent results of Ranke behooves us to recommend the early operation, while the value of the expectant treatment does not seem to be sufficiently proven.

The surgical treatment consists in excision of the affected parts through the healthy tissues and the immediate cauterization of the edges with the thermocautery.

The medical treatment is restricted to sponging and flushing with antiseptic remedies, of which hydrogen peroxide (2 per cent.) is comparatively the most effective. In addition pencilling with tincture of iodine or silver nitrate (10 per cent.) with a local tamponade of iodoform gauze may be tried. Recently, the daily brushing with a one per cent. solution of pyoktanin has been warmly recommended (Poljakoff).

THRUSH (SOOR OR SPRUE)

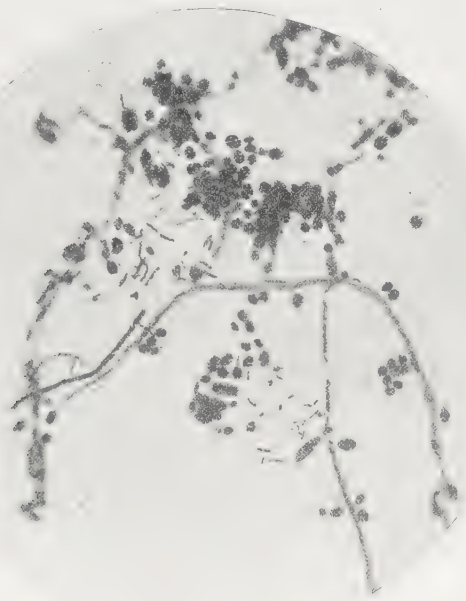
Thrush is a local disease of the oral mucous membrane produced by the growth of a specific fungus (thrush fungus).

The disease was recognized and described by the Hippocratic writers under the general term aphthæ, but the nature of the disease was obscure until the deposits of the specific fungus were described by Langenbeck, Berg, and Gruby in 1840, and at once the parasitic nature of the disease was established.

The name "soor" used by the Germans is derived from the low Saxon word *sôr*, that is, dry, parched, which probably arose from the fact that the thrush fungus grows with preference on a dry or withered mucous membrane. This stomatitis has been well known among the people for ages, on account of its characteristic appearance and there is scarcely a single disease which has so many appellations as thrush. At least 25 different names for this affection appear in the literature.

Pathogenesis, Local Symptoms.—The onset of thrush is manifested by the appearance of small round white spots upon the mucous membrane of the mouth. The preferable seat of onset is the tongue

Fig. 7.



Thrush. Smear from the mouth. Deposits scraped off, washed and allowed to remain in the water for a short time. Stained according to Gram. Enlarged 600 diameters.

(especially the fore part), the cheek, and the gums (see Plate 34). When an attempt at mechanical removal is made, we become convinced that it adheres more or less firmly to the base, a very important differential sign from the similar case in floccules which often adhere to the sides of the cheek in infants after vomiting. Furthermore, on inspecting the mouth, we observe objective signs of a simple stomatitis which is revealed by a redness and tenderness of the mucous membrane and which usually precedes the specific affection.

A microscopical examination of a mass which has been removed, shows that it is composed chiefly of a dense fungous growth, which consists of jointed filaments and shining gonidia, the thrush fungus (Fig. 7), also epithelial cells and a few leucocytes. If this fungus is grown on the

proper culture medium (obliquely hardened gelatin), it will be observed that the growth of the fungus is not restricted to the surface, but has a marked tendency to grow deeper, as an innumerable number of filaments from the superficial colonies have penetrated the whole depth of the culture medium.

A similar property, though in less degree, is shown by the fungus growing on the living mucous membrane; consequently, the deposit cannot be removed without leaving some of the growth and without removing some tissue cells, because the growth is implanted deeply with many radicles. The whole thickness of the epithelium to the connective tissue seems to be perforated by the fungous masses.

Gradually, the process extends over a greater surface. The mucous membrane of the lips, the tongue, the palate, and the cheek becomes covered with numerous white spots. The intervening free mucous membrane is inflamed, especially is this noticeable on the tongue, where the swollen papillæ protude like pegs from the centre of an area encircled by the fungous growth.

The adjoining colonies usually become confluent and consequently the surface of the tongue and cheek appear to be overspread by a white coat. At this stage the membranes do not adhere so closely; their removal is very easy, and they drop off piecemeal, spontaneously. A change in the color of the deposits is also manifest. They become yellowish or dirty brown, which discoloration is mostly due to superficial hæmorrhages.

The exfoliation of the thrush membranes is the consequence of a strong proliferation of the new epithelium under the deposit, and no visible alteration of the mucous membrane remains after healing.

Anatomy.—As a rule, the growth of the thrush fungus remains restricted to the epithelium; the epithelial cells appear loosened and perforated by the mycelia. The filaments may, however, grow into the connective tissue and even penetrate the walls of the blood vessels (Wagner). In this manner and by this route, as well as through the ulceration of the mucous membrane, the thrush fungus may gain entrance to the circulation.

Localization.—In most cases the growth of the thrush fungus is restricted to the cavity of the mouth; not infrequently, it may extend to the bordering structures of the throat, the œsophagus and also the nasopharynx (descending thrush).

The observation that the growth is limited by the cardia, the choanæ and the vocal cords led Berg to propound the hypothesis that the vegetation of the thrush fungus necessitates the presence of squamous epithelium (Berg's law). Although this singular behavior is certainly true as a rule, there are exceptions. To these belong the growth of the fungus on the turbinated bodies and nasal septum in infants afflicted

with wolf's throat; also the discovery of Heller, who found that the thrush filaments may penetrate the intact cylindrical epithelium of the trachea. Usually, the thrush fungus begins its growth in the mouth and from this place spreads to neighboring organs. It is possible, however, for thrush to grow primarily on the tonsils, and the mouth be free from the disease. Then the tonsils may be covered by a white pseudomembrane and thus simulate genuine diphtheria (sprue-diphtheria).

General Symptoms.—The local changes are introduced by and attended by other symptoms. The infants drink less greedily; in fact, on account of the pain, food may be ingested in very small quantities. The body weight falls, which may be the result of a diarrhœa which usually attends the disease. Slight fever and vomiting are often present. If the thrush is propagated to the upper air-passages, hoarseness and a slight cough may supervene.

Course.—Thrush usually lasts for a short time only. Under proper treatment it disappears in one or two days and does not return. There are obstinate cases, however, which resist therapeutic measures for many days. Occasionally, thrush disappears temporarily to return again, perhaps during the night, as luxuriantly as ever. The infants in these cases are debilitated, atrophic or cachectic, in whom thrush is only a complication of another severe disease (cachectic thrush). The appearance of thrush is always a serious symptom in these cases, because it often announces a general decline of the vitality. In these isolated cases we see thrush descend to the deeper parts of the throat and the necropsy reveals an extensive thrush of the œsophagus (see Plate 36).

Thrush does not always remain a local infection; in rare cases the fungus may produce a specific general infection of the organism. This leads to a discussion of the severest complications of the "sprue-disease," to which an anatomical rather than a clinical interest must be attached, as they are recognized only after death, and do not manifest any characteristic symptoms during life.

The tendency of the thrush fungus to induce genuine metastases belongs above all to this class. These were first described by Zenker and Ribbert,—each reported a case of multiple cerebral abscesses; later, Schmorl, Guidi and Pineau saw embolic abscesses of the kidneys, lungs, and spleen. Recently, Heubner observed a similar case in an infant, in which during life a probable diagnosis of "general infection by thrush" could be made. The formation of metastases, as well as the general infection, is readily explained by the ability of the thrush fungus to penetrate into the blood vessels (see Plate 36).

The cases of thrush-sepsis may be more frequent than is usually assumed. Systematic puncture of the heart immediately after death at the clinic of Escherich many times revealed the fungus in the blood of the heart. The researches of Stooss have demonstrated that the

thrush fungus in its passage is accompanied by other microörganisms, especially the pyogenic streptococci and staphylococci, which are always abundantly present in the thrush membranes and in the deeper layer of the tissues into which the fungus has penetrated. Hence, it is not improbable that a mixed infection holds an important rôle in the formation of metastases and general sepsis.

The pyogenic properties of the thrush fungus, proven by experiment on animals, furthermore explain the finding of this microörganism as a causative agent in purulent otitis media. The infection of the middle ear probably occurs through the short passage of the Eustachian tube.

While the severe septic complications perhaps belong to the rarities of medicine and hence do not incite a great practical interest, it is expedient to discuss a group of phenomena accompanying the local process and forming a part of the clinical syndrome.

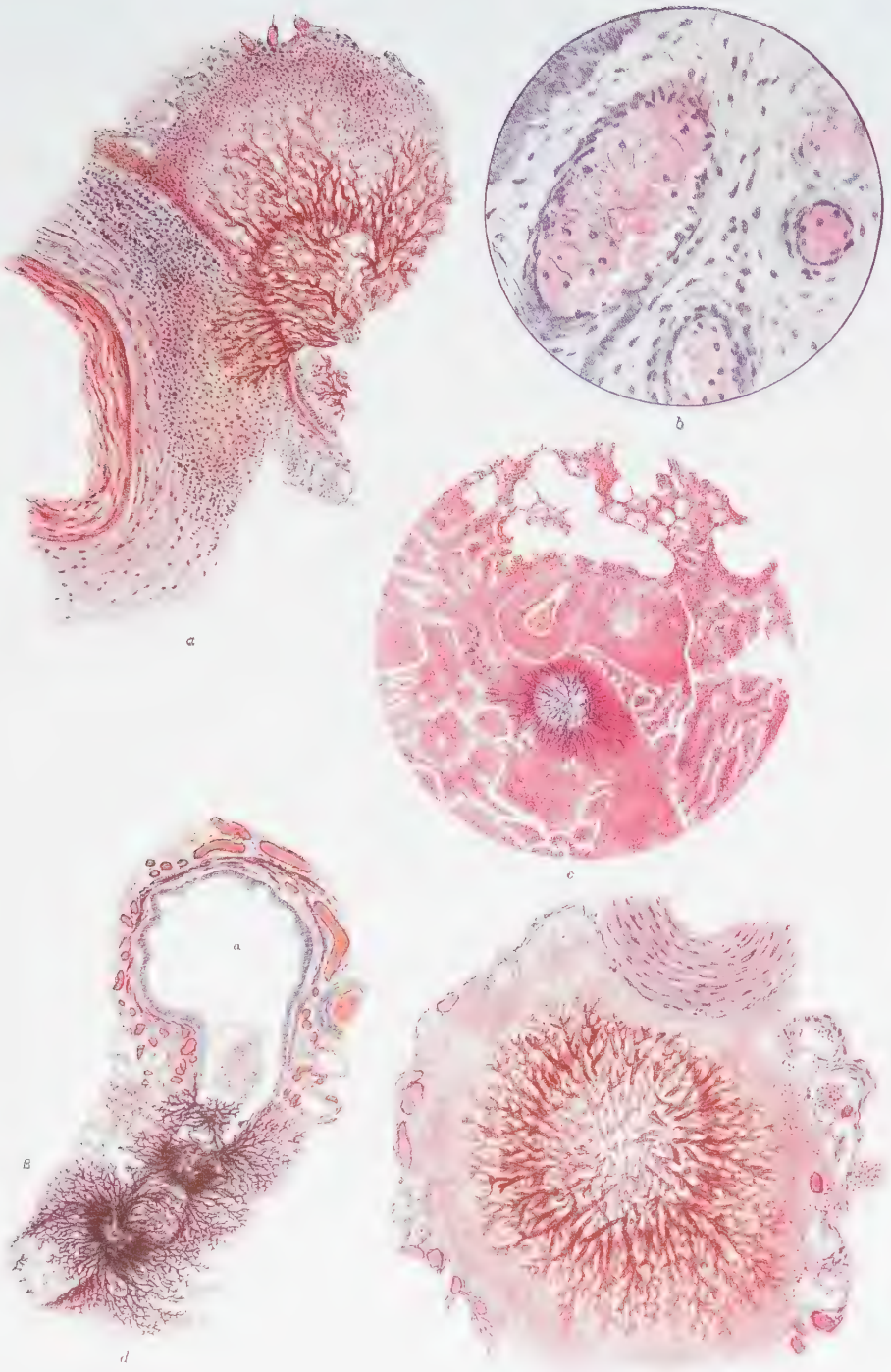
In the first place the intestinal symptoms already mentioned should be considered, which arise mostly in the form of an intestinal catarrh of varying severity. The question whether thrush is the result or the cause of this intestinal disease is not conclusively decided. Experience teaches that a catarrh of the intestine, or at least a dyspepsia, precedes the outbreak of thrush and that perfectly healthy infants, as a rule, do not become affected with thrush. Although this rule certainly has its exceptions, especially in breast-fed infants, it is not always easy to draw a line between the physiological and pathological variations of the stools; still it must be asserted that infants suffering with some intestinal disorder have an increased susceptibility to the local invasion of thrush.

One thing is certain, namely, that thrush may be the direct cause of complicating intestinal diseases. Masses of the fungus are swallowed in large numbers in this stomatitis, which find favorable conditions for vegetation in the acid condition of the intestinal contents and can give rise to a specific affection. The infection in some cases may take the form of a severe febrile enteritis, which only yields after the cure of the local process. The bacteriological aspect of the stools in such cases teems with elements of the thrush fungus and the normal flora occasionally disappear almost completely.

The diarrhœa which so often results from thrush is the cause of another complication, namely, intertrigo. It is not improbable that the masses of fungi in the stools may participate in the production of this dermal inflammation, which develops further on the buttocks. The fungi as a rule can be easily found in large numbers at that place.

A third complication may ensue: this is the frequent occurrence of Bednar's ulcers of the palate in infants afflicted with thrush. Their appearance is brought about by the efforts of swabbing the mouth for the purpose of removing the patches. These manipulations lead to lesions of the vulnerable mucous membrane of the mouth.

PLATE 36.



THRUSH FUNGUS IN THE TISSUES.

a. Growth in the walls of a branch of the pulmonary artery with surrounding round-cell infiltration.

b. Thrush in the submucosa of the œsophagus extending into a blood-vessel.

c. Thrush in the lungs. Growth of the fungus in one of the bronchioles in a pneumonic affected tissue.

d. Thrush in the ductus alveolaris with extension into the surrounding tissue. (*α*. Cross-section through a bronchiolus. *β*. Extension into an alveolar duct.)

e. Extension of the fungus from a bronchiolus into the surrounding tissue without small-cell infiltration. Vessel-wall remains free.

Occurrence.—Thrush is peculiar to the age of infancy, and, furthermore, infants in the first two months of life are attacked most frequently. It is remarkable and gratifying to find that thrush is becoming more infrequent, and that the severe cases particularly, which were so constituted as to stamp thrush as one of the most dreaded diseases, even the very scourge of maternity hospitals and foundling homes, now belong to the group of clinical rarities. This is a triumph of modern hygiene and not the least good result from the discontinuance of the excessive washings of the mouth in healthy infants.

In older children thrush as a primary disease is rare. As a rule, the thrush fungus implants itself, as in adults, only during the course of some other severe disease which impairs the nutritive condition (as tuberculosis, meningitis, typhoid). Once only I found in a vigorous boy a white pultaceous deposit confined to both tonsils, which resembled diphtheria, but which was composed of thrush masses (sprue-diphtheroid).

The increased predisposition of infants to thrush is explained above all by the dryness of the mouth and occasional acid reaction of the mucous membrane. Both conditions favor the implantation and vegetation of the thrush fungus as experience has shown; while the normal alkaline saliva has an inhibitory effect on the growth of the fungus. Furthermore, it must be mentioned, the organs of the mouth in the infant remain mostly in a state of rest. The important act of self-cleansing the mouth, therefore, is deficient, a function which in later life is a very essential task.

The alimentation, whether natural or artificial, has no substantial influence. We find breast-fed infants, as well as those artificially fed on the bottle, attacked by thrush. In the former, however, the disease as a rule runs a more favorable course, which must be attributed to the greater power of resistance generally present in breast-fed infants.

Beside the gastro-enteric disease, a simple stomatitis, often the result of too frequent sponging of the mouth, is a predisposing factor. Careful experiments have demonstrated that the direct or indirect transference of the thrush fungus upon the completely intact mucous membrane of healthy infants never succeeds (Epstein, Soltmann).

Etiology.—The specificity of the thrush stomatitis is definitely established since the discovery of the fungus in the deposits on the mucous membrane. The opportunities for infection are numerous on account of the abundance of the fungus. Almost from every stool of the breast-fed infant, from the mammillæ of the nursing woman, and from cow's milk, the thrush fungus may be cultivated. The fungus is relatively less frequent in the atmosphere, so that one must think of a direct infection by contact in the first place. The fungus may readily be conveyed from one to the other, through filthy "pacifiers" and rubber nipples, through the act of drinking from the breast, but especially

through the objectionable washing of the mouth. The fungus is a common inhabitant of the healthy mouth, where it leads a saprophytic existence without inducing a specific disease.

The classification of the thrush fungus into the plant life has offered great difficulties for many years, and has lead to a variety of polemical discussions. According to the exhaustive researches of Plaut, the fungus, which belongs to the order of hyphomycetes, stands nearest to *monilia candida*. Its pure culture is most easily obtained on an acid solid medium, by which it is possible to isolate the fungus from its attending bacteria. Very remarkable is the great tendency of the thrush fungus to produce varieties (variety with small gonidia, liquefying variety, etc.). In the future it may be possible to establish the relationship of these varieties to the different forms of the disease.

The **diagnosis** seldom presents any difficulties. The differentiation of the colonies of the fungus from coagula of milk has already been explained. The surest proof in every case is offered by a microscopical examination, which alone can indicate the correct condition in those deposits confined to the tonsils, which resemble diphtheria, as previously mentioned.

The **prognosis** of this stomatitis depends on the condition of the afflicted infant. In a healthy vigorous nursling thrush disappears certainly in a few days, without causing any serious disturbance. In the debilitated, the premature, or infants weakened by grave diseases, thrush may threaten life, because it interferes with the ingestion of food, which is already lessened, and because the resulting diarrhœa and complications hasten the decline of the vitality.

If the described *prophylactic regulations* are accurately carried out in the modern hygiene of infancy, it is possible to be entirely exempt from the thrush plague. The principle regulations are: desistance from mouth washing in the healthy infant, use of its own utensils, boiling the nipples, avoidance of unclean "soothers," removal of soiled diapers, and keeping the mother's nipples clean. It follows, therefore, that thrush is entirely absent from asylums conducted under model hygienic regulations.

Treatment.—Boric acid as a specific medicament against thrush continues to hold first place. Kehrer has shown that this drug has the power to inhibit the growth of the fungus. In order to avoid the dangerous and inefficient washing of the mouth, Escherich introduced the ingenious boric acid teat. This has the shape of the old sugar-teat, and consists of a compress of sterilized cotton, which has been dipped in finely pulverized boric acid and then wrapped in gauze or fine batiste, forming a small ball, from a strawberry to a hazel-nut in size. Before its use, in order that it may be taken with relish, the teat may be dipped in a 0.01 per cent. solution of saccharin. The boric acid teat must be

made to fit the mouth, and must not be too large or too small. It must be often administered and must be shielded from contamination of every kind.

Most infants take this teat with relish, make vigorous sucking and chewing movements, and gradually dissolve the powder with the saliva. It is obvious, that this singular method of treatment is innoxious and that in this way a thorough mechanical and antimycotic cleanliness of the mouth may be obtained. If the thrush has just begun, or has not extended very far, the use of the teat will cause its disappearance in 24 hours. In obstinate thrush or in infants by whom the teat is refused, the infant should be allowed to suck a small brush dipped in nitrate of silver glycerin (Argen. nitrat. 2.0, glycerin 20.0, aqua destill., 80.0—Concetti) or a 2 per cent. solution of silver nitrate may be applied.

ULCERA PTERYGOIDEA (BEDNAR'S APHTHÆ)

The above disease of the mouth was first described by Bednar and classified under the general name aphthæ. Hence arose the designation "Bednar's Aphthæ" which is generally used even to-day.

Epstein, to whom we owe an excellent description of the disease, suggested the appropriate name "ulceration of the palatine angles" (gaumeneckengeschwüre), which we will use on account of the confusion resulting from the term aphthæ.

The disease attacks exclusively the newborn and infants in the first weeks of life.

Nature, Localization, Symptoms.—The process is mostly bilateral and symmetrical, and is manifested by the appearance of circumscribed superficial ulcers in the region of the hamuli pterygoidei. The ulcers are about the size of a pea or a bean and are encircled by a red areola. They are covered by a yellowish white mass which adheres firmly to its base; hence bleeding results on their removal. The ulcers are painful, and, therefore, cause an inability to nurse on the part of the infant.

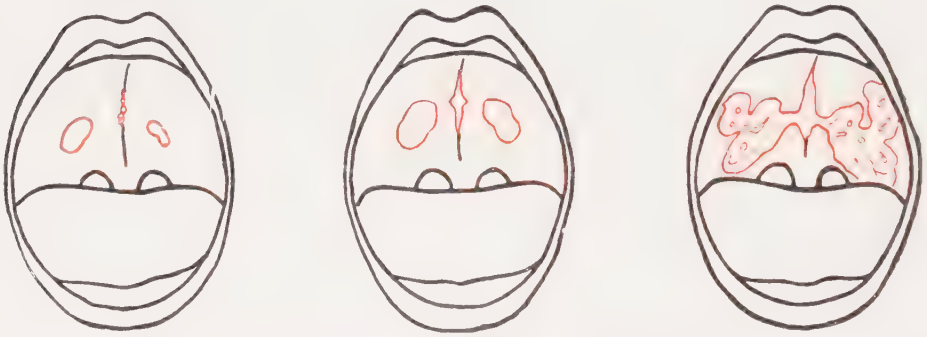
Course.—After a few days the necrotic masses are spontaneously exfoliated; the grayish red floor of the ulcer is denuded but is rapidly covered by new epithelium from the periphery.

Both in regard to localization and the course of the disease, deviations from the described typical picture may occur. Sometimes the ulcer may be found on one side only while the other side is intact. Occasionally two lesions may be joined by a bridge giving the ulceration the appearance of Fig. 8. The pathogenesis of the disease makes it possible for the ulcers to occur on other parts of the mouth. Thus we see exceptionally ulcerations appear on the raphe of the hard palate, in the places where in young infants the epithelial pearls are to be found. These ulcers, in correspondence to the anatomical structures of the base, have the form of a rye grain or a lancet.

In some cases typical ulcers at the angles of the palate, and, in the middle line an ulceration of the raphe may develop. All of these lesions may become confluent by extension of the necrotic process so that the greater part of the palate may appear covered by the yellowish white deposit. In consequence of its symmetrical arrangement around the middle line, the deposit often assumes the strange shape of a butterfly whose body is formed by the central ulcer of the raphe, while the wings are represented by the lateral ulcers. These are the forms which are known as pseudodiphtheria, although they show very little resemblance to the clinical picture of diphtheria (see Fig. 8c).

Quite different are those cases in which the signs of a congenital stenosis of the upper air-passages precede the formation of the erosions. The causal connection of both these phenomena has recently been announced by Breceli (from Pfaundler's Clinic). A mistake in diagnosis in these cases is conceivable.

Fig. 8a. Fig. 8b. Fig. 8c.



Schematic representation of Bednar's ulcers and the median ulcer of the raphe (a and b). Confluence of the ulcers leads to pseudodiphtheria (c).

The lymph and blood vessels are opened by this ulcerative process and since pyogenic agents are present in large numbers, the opportunity for a general infection is offered. These conditions attending the evolution of Bednar's ulcers may lead to a severe and serious disease, especially in debilitated and atrophic infants.

The *morbid anatomy* of this disease, according to Fränkel and Eppinger, consists in mycotic necrosis of the epithelium. The microscopical examination of the removed deposits of the ulcers shows that it is made up of desquamated epithelium, leucocytes and innumerable microörganisms. In smear preparations, as well as in the culture, streptococci predominate among the bacteria. These probably play a part in the etiology of the disease; certainly they may cause general sepsis.

Pathogenesis, Etiology.—Why do these ulcers always occur at the angle of the palate in the region of the hamuli pterygoidei? The most plausible answer to this question is that the mucous membrane of the palate there is tensely stretched and, consequently, can easily become

the seat of superficial lesions during the act of nursing or washing the mouth. Epstein, particularly, attributes the local injuries to the forcible and routine washing the mouth, which, unfortunately even to-day is commonly practiced by mothers and nurses. In favor of this argues the experience that Bednar's aphthæ occurs principally in infants in whom diligent wiping of the mouth has been done, while the disease has become infrequent since this practice has been discontinued. The disease follows thrush with extraordinary frequency. It is rational to assume that the appearance of the deposits in the mouth induces the mother to cleanse the mouth vigorously. In favor of the mechanical theory is the fact that the disease occurs on the prominent parts of the mouth, such as the ridge of the palate and the epithelial pearls.

Against this theory the opponents contend that Bednar's ulcers do not usually appear over the hamuli pterygoidei, but rather to the inner side and also on the horizontal plate of the palate bone. Extensive investigations of E. Fränkel have shown that the mucosa at that place does not show any difference in regard to thickness; neither is its tension marked, for the mucous membrane possesses a distinct mobility which is uninfluenced by crying or sucking. The characteristic localization, as explained by Fränkel, is that at this place in the mouth there is a kind of dead point which favors the accumulation and implantation of microörganisms.

For the **prophylaxis** of pterygoid ulcers, desistance from washing the mouth is the chief rule. In the second place, a rational treatment for thrush must be instituted, because it frequently favors the development of the ulceration, especially when the ordinary method of washing the mouth is used.

Treatment.—If a 2 per cent. solution of silver nitrate is daily applied to the eroded place, the disease is readily controlled and the ulcers heal rapidly. If the infants show a disinclination to take food on account of pain, the application of a 1 per cent. solution of cocaine before feeding, or the insufflation of orthoform powder will give relief.

STOMATITIS GONORRHOICA NEONATORUM

A series of cases have been reported which make it probable that the implantation of Neisser's gonococcus on the mucous membrane of the mouth may lead to a specific stomatitis (Rosinski, Leyden, Kast).

On certain places of the oral mucous membrane,—usually in the angles of the palate, along the raphe and in the gingivolabial folds, a whitish, elevated exudate appears. This arises without a preceding inflammatory redness, has a rough surface and gradually changes to a yellowish discoloration. The process is confined to the mucous membrane and heals spontaneously without scar formation in a few days. The **prognosis,** therefore, is entirely favorable.

The possibility of a specific infection of the mouth in the newborn is given in its passage at birth through an infected genital canal and later on by contact. In the epithelial layers numerous microorganisms are found which have been pronounced gonococci from their morphological qualities. However, as the cocci in stained sections have never been discovered intracellular and since cultures have not certainly been successful, these findings must be cautiously received, especially since bacteria resembling gonococci are often found in the flora of the mouth in infants. I have often found similar bacteria in the deposits of Bednar's aphthæ and in tonsillar exudates.

PERLÈCHE (EPSTEIN'S "FAULE ECKEN")

The ulceration at the angle of the mouth in children (Fig. 9) was recently accorded an exhaustive description by Epstein (1900). The disease has been known by the laity for a long time under various names (bad corners of the mouth, foul angles, etc.), and is an ulcerative process of the commissure of the lips. The affection was described earlier (1886) in the French literature under the name "perlèche."

Symptoms.—The disease is limited exclusively to the angles of the mouth and begins by the appearance of a circumscribed redness with uneven surface. The roughness of the affected area is produced by numerous grooves which have a characteristic feature, namely, they radiate from the angles of the mouth, whereby the affection suggests the shape of a fan. The skin of this region has a darker shade and is brownish in color. As a consequence of the desquamating epithelium the surface has a rough appearance. As a rule, both angles of the mouth are symmetrically implicated. When the angles are stretched by opening the mouth, moist fissures, sometimes covered with an exudate, may be seen. These are very painful and bleed easily when they are pulled apart.

In order to mitigate the local distress and to cool the burning sensation, the children are accustomed to lick the affected parts (hence the French name "perlèche" from *pour lècher*) and to suck the air to the corners of the mouth. No other trouble is caused by this harmless affection. It heals spontaneously, as a rule, without scar formation. Occasionally, a white, shining surface temporarily remains at the inflamed area. Of course, the process may sometimes be protracted if the proper medical treatment is not employed.

Diagnosis.—Perlèche has a diagnostic significance in that it may be mistaken for syphilitic plaques. It is distinguished from the latter disease by its strict localization; perlèche, moreover, does not extend to the inner surface of the cheek and has a benign appearance and does not lead to a deep ulceration of the tissues. In doubtful instances the history of the case and a general examination of the body must decide.

Etiology.—The disease is found chiefly among children at the school-going age. This fact, in connection with the observation of epidemics among the members of the family, supports the hypothesis that the disease has an infectious origin. Lemaistre, to whom we owe the first accurate description of the disease, charges an anaërobic streptococcus as being the specific excitant, since it is regularly found in the epithelium of the affected parts.

Fig. 9.

Perlèche in a child two and one-half years of age.

For the **prophylaxis**, the avoidance of direct or indirect contact with the specifically infected area (by kissing, drinking or eating from common vessels) holds first place.

Local **treatment** is confined to daily application to affected parts of tincture of iodine, balsam of Peru, or the tincture of rhatany. A drying treatment with dermatol or orthoform powder will also succeed.

IV. SECONDARY AFFECTIONS OF THE MOUTH

As a complication of severe and protracted diseases, as well as a consequence of infectious diseases, secondary diseases of the oral cavity may arise. This observation suggests that the mucous membrane

during the course of these diseases acquires a diminished power of resistance. The following must be considered as predisposing causes: the lessened power of self-cleansing due to the relative dryness of the mouth, as in fevers; the insufficient care of the mouth during the disease; and the cachectic condition of the mucous membrane which forms a part of the general depression.

In considering the several forms of stomatitis, we have already noticed that ulcerative stomatitis (cachectic) and especially noma are frequent complications of the infectious diseases. Measles, especially, as experience has taught, seems to predispose to these and other diseases of the mouth. Frequently we observe in measles an inflammation of the mouth which shows a marked resemblance to maculofibrinous stomatitis. The white efflorescences arise in various parts of the mouth, more frequently on the gums in front, on the inner side of the lips, and on the mucous membrane of the cheek. Yet its tendency to lead to a superficial necrosis and ulceration indicates that it must be separated from the maculofibrinous stomatitis and must be regarded as a distinct affection (necrotic stomatitis after measles).

Likewise during the course of scarlet fever, diphtheria and typhoid, secondary and generally ulcerative processes may arise, which lengthen convalescence by their painfulness and by hindering the intake of food. (See the chapters describing these diseases, also the changes of the mucous membrane in scurvy and in syphilis.)

The mouth of infants afflicted with sepsis is often the seat of secondary morbid changes. Besides the appearance of a septic enanthem and punctiform subepithelial hæmorrhages, which are identical in their nature with similar changes on the skin and appear most plainly on the palate or mucous membrane of the cheek, the oral cavity in septicæmia is occasionally the seat of an extensive secondary process, which was observed at first by Epstein, and described as pseudodiphtheria of septic origin. Further observations of this rare disease have been made in Pfaundler's Clinic (Breceli).

The small ulcers of the oral mucous membrane at the onset rapidly enlarge and spread toward the organs of the throat. On account of this localization and on account of the grayish yellow discoloration of the exudate, the disease suggests diphtheria, especially since in certain cases a genuine fibrinous exudate, and tough elastic, and recurring membranes are formed in the mucous membranes of the throat, which show a tendency to invade the respiratory tract. The local process as a rule does not arise until a few days before death, but it must be recognized so that the erroneous diagnosis of diphtheria be not made.

Recently, Swoboda described a fatal disease of the mouth under the name "gangrenous inflammation of the dental germs in early infancy," *

* Identical with the disease described by Klementkowsky as "Osteogingivitis gangrænosa neonatorum."

which may occur during the course of some septic disease in the newborn or very young infant. The characteristic symptom of this rather rare disease (six observed cases) is the gangrenous disintegration of the gums and the subsequent avulsion of the crowns of the teeth in infants who are still very far removed from the dentition period.

Finally, the secondary processes of the mucous membrane which occur in children ill with cardiac or hæmorrhagic disease must be remembered. Here isolated grayish yellow plaques make their appearance and arise from minute embolic hæmorrhages of the mucous membrane. The destruction of the epithelial cover leaves a minute loss of substance which becomes covered with a white deposit. These plaques have a certain resemblance with the eruptions of maculofibrinous stomatitis, but are distinguished from these particularly by the transition to superficial necrosis and finally by the fact that only one is present.

V. ANOMALIES OF THE TONGUE AND THE FRENUM

It is a fact well known to the physician, as well as the laity, that the surface of the tongue at the onset and during the course of various diseases becomes covered by a whitish coat. The *coated tongue* played a very prominent rôle in older medicine. As the tongue forms the first portion of the digestive canal, it was easily deduced that changes on the surface were caused by some digestive disturbance. The older physicians even entertained the fantastic conception that the arrangement of the deposit in the surface of the tongue could be utilized to locate the digestive disturbance.

While no one to-day would attempt to solve the problems of disease by the shape and character of the coat on the tongue, its significance should by no means be entirely discarded. On account of an inflammation of its tissues the tongue assumes a characteristic appearance in certain infectious diseases, as scarlet fever and typhoid, which change becomes a valuable aid in diagnosis. In fevers of an ephemeral nature, in which other local symptoms are absent, a very marked coating of the tongue must not only be regarded as an attendant of the fever, but should also raise the suspicion that the gastro-enteric tract is the source of the fever. An intense acetone odor emanates from the mouth during its inspection and a more careful inquiry will show that some error in diet has been made. But in general, the coating of the tongue as a diagnosic aid has correctly assumed a subordinate position.

A more important change in the surface of the tongue is that which is known under the name of *geographical tongue* (lingua geographica). This name, as well as the synonyms, annulus migrans, glossitis areata exfoliativa, clearly express the remarkable appearance and partially the nature of this disease. At the outset it should be stated that this singular anomaly of the tongue in childhood is different from that form

described under the name of leucoplakia or psoriasis linguæ, but nevertheless is frequently placed in the same class as the latter affection.

On the surface of the tongue, most commonly at the edge, a grayish white spot appears, which rapidly enlarges. Its growth is usually in the direction of the dorsum of the tongue, rarely toward the base. At the same time the grayish white color in the centre disappears and a red discoloration takes its place, which seems encircled by the peripheral remains of the white deposit. The grayish white border moves onward in concentric rings, reaches the middle of the tongue and occasionally passes over it. When several such irregular figures coalesce, the characteristic geographical map is formed. In the further progress of the disease, the reddish discoloration of the encircled areas becomes pale, so that only the white stripes remain. The rest of the tongue may be coated.

These changes, which never lead to ulceration, remain localized on the surface of the tongue, as a rule, and run a chronic course with frequent and rapid changes of the formations. The grayish discolored parts consist anatomically of circumscribed epithelial hyperplastic areas, while the subsequent red color is produced by the exfoliation of the epithelium and the epitheloid processes of the filiform papillæ and by the projection of the fungiform papillæ. The process advances with an infiltration and exudation and is confined chiefly to the upper layers of the epithelium.

The disease attacks mostly children between the first and fourth year of age. The disease according to all appearances seems to be independent of the presence of teeth, since it has been observed in toothless infants.

The geographical tongue must be regarded as a partial phenomenon of a constitutional anomaly. Bohm declared that he found the geographical tongue most frequently in scrofulous children, but the investigations of Carow recently tend to throw some doubt on this observation. Czerny saw the disease especially frequent in children afflicted with the exudative diathesis.

The geographical tongue certainly has nothing to do with syphilis, but the latter disease may have to be considered in differential diagnosis, as the secondary erythematous syphilide of the tongue may have a similar appearance on the surface. The syphilitic eruptions do not migrate, mostly ulcerate and yield promptly to antisyphilitic treatment.

The possibility of a parasitic nature of the disease, in analogy to similar skin diseases has been considered, without any definite proof however.

The medical treatment of the affection has no special object and is without permanent result. For cosmetic reasons the treatment recommended by Seiffert may be used. This consists in the application of a strong solution of chromic acid for a few minutes and the subsequent washing with a solution of aluminum acetate.

A typical change of the tongue is often observed in children whose mouth contains carious teeth. The tongue suggests, in a measure, the alterations which we have learned in ulcerative stomatitis. The edge of the tongue is indented in places, which is really an imprint of the teeth. There is an inflammation of the margin of the tongue from the pressure and irritation of the sharp edges of the carious teeth. Occasionally a very painful ulceration may arise from this cause, but it heals promptly on removal of the cause.

Under the name *erythematous inflammation of the side of the tongue* in infants (glossitis marginalis erythematosa), Wertheimer describes an inflammation which as a rule is characterized by a marked redness and desquamation of the epithelium. The affection attacks young, artificially fed infants and is attributed to the irritation from the act of nursing. This process, which must be considered almost a physiological appearance, offers no further interest and on account of its harmlessness needs no treatment.

A greater interest is excited by those anomalies of the tongue which rest in an abnormal increase of its volume. *Macroglossia* is rare in children as a distinct disease (lymphangioma linguæ) but frequent as an integral part of the symptom-complex in other diseases. In idiocy, myxœdema, mongolism and acromegaly the enlarged tongue belongs to the syndrome of abnormalities which characterize these diseases.

The enlargement of the tongue in these diseases is due to an increase in the interstitial tissue. The organ in consequence of its increased diameter finds no place in the mouth and hence the anterior part protrudes. In the very nature of the case, this voluminous tongue is disturbed in its function in the act of nursing, as well as later in speaking; and its constant contact with the teeth and the atmosphere predisposes to inflammatory processes, which often result in a fissured and eroded condition if the tongue (*Lingua dessicata*).

The treatment of macroglossia lies in the province of surgery and consists in the excision of a wedge-shaped piece of the anterior part of the tongue and subsequent suture. The tongue enlargement resulting from diseases of the thyroid is favorably influenced by a specific organotherapy.

A few words concerning the inflammatory affection of the sublingual tissues must added. This condition was first described by Holthouse as *subglossitis* and later by Henoch as "inflammation of the floor of the mouth." In these cases there is a phlegmonous inflammation of the submucous connective tissue at the border of the tongue, which spreads to the adjacent tissues and is attended by high fever and great local distress (enormous swelling, elevation of the tongue, salivation). It terminates with a discharge of pus under the tongue or externally.

The etiology was unknown to the observers. Nothing is known concerning the avenues by which the pyogenic agents enter.

The *frenum* is the seat of secondary morbid conditions more often than the tongue itself. The most common is the sublingual ulceration in whooping-cough (see Plate 34). The formation of the ulcer begins in the middle or at the side of the frenum. In the course of the process the whole frenum becomes eroded and in its place a broad deep ulcer is seen, which is covered by a white exudate and surrounded by an elevated border. During the paroxysms of coughing the tongue is pressed and rubbed against the incisor teeth and this produces a decubitus ulcer of the frenum. A similar change of the frenum is sometimes seen as a complication of dentition in infants who are in possession of the lower central incisors only.

Of special interest is the neoplasm of the frenum which arises spontaneously and was first described by the Italian physicians under the name of *produzione sottolinguale* (Fede) or rather as *fibroma sublinguale* (Callari and Philippson). This affection has assumed a place in the German literature under the name of *Riga's disease*, although the priority belongs to Pandolfi (1875) and not to Riga (1880).

The disease attacks infants exclusively. Its geographical distribution is confined almost entirely to the southern provinces of Italy, but it has been observed in Venice, France and Austria.

The affection begins as a minute opaque whitish thickening in the frenulum. The growth rapidly increases in size and attains a diameter of about 1 cm. and shows in the centre a shining white area while the rest of the growth has a red appearance. The growth feels hard on palpation.

The histological examination of the little tumor shows that we have to deal with a fibroma. To this are added inflammatory processes, which are strictly limited to the apex of the growth. At this point a large number of leucocytes are found which are mostly of the eosinophilous type. The inflammation ultimately leads to a superficial necrosis of the fibroma, which is the cause of the whitish discoloration.

The disease causes no other disturbance. Its *treatment* is entirely surgical. Callari and Philippson regard the disease as an hereditary anomaly, a racial peculiarity, which is disseminated by heredity throughout the mentioned countries, and to which it is confined. An original relationship to dentition has not been definitely proven, but the presence of the lower central incisors standing alone seems to be a predisposing factor. Another explanation of the frequent occurrence of the disease in Italy is that the mothers in the southern provinces nurse their babies long after the dentition period. During the act of nursing, the teeth can produce a considerable mechanical irritation of the frenum and thereby cause the growth.

An abnormal condition of the frenum exists when it is too short or when it is inserted far forward to the tip of the tongue. This anomaly

very seldom causes any trouble, but in order to meet the urgent demands of the mother or midwife, it can easily be torn through with one blade of the scissors.

In rare cases the lower surface of the tongue is grown to the floor of the mouth. This abnormality is usually congenital and consists in an epithelial adhesion which can readily be separated mechanically.

VI. ANOMALIES OF DENTITION AND THE SHAPE OF THE TEETH

DIFFICULT DENTITION

The eruption of the teeth frequently occurs without causing the least distress to the infant. On the other hand, we also observe dentition preceded and attended by more or less definite symptoms, which point to a local or general disturbance of the infantile organism. Two extreme views in regard to the significance of these symptoms are extant: one maintains that dentition is purely a physiological phenomenon and does not induce any alteration in the feelings of the infant, and that any disturbance in health during dentition must be regarded as the symptoms of another disease coincident accidentally with the eruption of a tooth. The other view goes so far as to attribute severe morbid conditions (diarrhœa, convulsions) to the process of dentition. The latter view agrees with that of the older physicians, while the former is a blunt contradiction.

It is out of the question, that the presentation of the newer views to the public can have but a wholesome effect; for with the welcome conception "teething," much evil certainly has been accomplished, inasmuch as other febrile conditions, which had nothing to do with dentition (as disease of the middle ear, the intestine, the lung, etc.), received no attention as they were considered temporary and favorable symptoms of teething. Yet the newer view absolutely does not agree with practical experience. What else can the physician do but attribute the symptoms to dentition when, as is often the case, a perfectly normal infant during the eruption of a tooth becomes very irritable, languid and depressed; when its desire to drink is lessened; when the thermometer indicates febrile movement, when, furthermore, the local inspection of the mouth shows a slight redness and swelling over a growing tooth,—all symptoms rapidly disappearing with the eruption of the tooth,—and when a careful physical examination of other organs shows only normal conditions?

The peevish disposition, the languor, the restless sleep (starting in sleep), the lack of appetite, an elevation of temperature (especially in the evening) and a simple stomatitis attended with drooling,—these are the common symptoms which assist in building a disease under the name of "difficult dentition." In some cases, in addition, vomiting and diarrhœa occur. Moreover, the occurrence of genuine eclamptic

seizures has been observed in some infants during the period of teething and never afterward.

While no one would try to find an immediate causal connection between the eruption of a tooth on the one hand and diarrhœa on the other, these disturbances may be readily explained by the experience, that the tender infantile organism may react in manifold ways to a sensible irritation, and to these undoubtedly belongs a painful dentition.

Since these symptoms depend on dentition, they have a fleeting existence and do not, therefore, require any special treatment. While the lancing of the gums probably has no advantage, brushing of the affected parts with aneson in order to diminish the local pain may be recommended.

Varieties of Dentition.—The time and order of the eruption as well as the process itself, may show variations from the normal. Occasionally infants are born with teeth (*dentitio præcox*), which generally drop out spontaneously or as a consequence of local inflammation. This freak of nature belongs to the rare curiosities and, therefore, possesses no practical importance. On the other hand, *dentitio tarda*, the late eruption of teeth, is important, since it is the most frequent symptom of rachitis. The first milk teeth may appear at the close of the first year or at the beginning of the second and not at six or eight months. If the rachitis arises late, the first incisors may appear at the proper time, but a long interval follows and the length of dentition is prolonged far beyond the normal. This delay in dentition is even more characteristic of infantile myxœdema, a disease which is closely related to rickets.

A variation in the sequence of dentition is most commonly associated with rickets. Occasionally, the upper incisors appear before the lower; sometimes the upper lateral incisors appear first; very rarely the canines pierce the gums before the molars.

Changes in the Shape of the Teeth as a Symptom of Certain General Diseases.—It is obvious that severe diseased conditions, which advance with a profound disturbance of nutrition, may affect the substance of the teeth, particularly when the osseous system forms the chief tissue affected. It follows that in the course of rachitis, or as a feature of a post-rachitic process various abnormalities in the shape of the teeth may be observed. Even excepting the fact that the abnormal growth of the jaw bones causes a crowding of the teeth in rachitic children, the permanent incisors in individuals who suffered from rickets previously, are subject to ridges, grooves, and erosions soon after their eruption. This deficient development of the permanent teeth probably depends on changes produced in the dental germ by the rachitic process during the first years of life.

The so-called *rachitic teeth* are by no means a reliable sign of a previous rickets, as there are persons whose extremities show a former

rachitis and yet possess faultless teeth. Local diseases of the teeth may produce imperfections in persons who never had rickets. These deformities of the teeth then, can be utilized as a corroborative sign of a previous rachitis only when other evidences of this disease are present.

Hutchinson's teeth, to which a special diagnostic value in hereditary syphilis has been attributed by that author (Hutchinson's triad), are similar in their nature, but differ in form and localization. The deformity consists in a semilunar excavation of both upper central incisors. The diagnostic value of this anomaly has been shattered in a great measure by very careful observers for reasons similar to those mentioned above.

It is the special merit of Neumann to have emphasized the relationship of circular caries and the circular adherent deposits at the

Fig. 10.

Hutchinson's teeth in a ten-year-old boy.

Fig. 11.

Circular caries of the upper incisors in a four-year-old boy with tuberculosis of the bronchial glands.

neck of the milk teeth to scrophulo-tuberculosis. Circular caries, which may affect the tooth at any time after its eruption, is located immediately at the junction of the tooth and gum, and, therefore, attacks that part of the tooth nearest to the gum. This caries is preceded by a greenish or brownish discoloration of that part of the tooth, which, bow-shaped, follows the line of the gum at first, but soon becomes larger. The onset of the caries is revealed by the loss of the shining surface and in place of this a rough chalky appearance is noticed, which is easily seen when the tooth is dry. Then the enamel crumbles off and when the dentine is exposed, the caries rapidly penetrates the tooth at its neck and leaves a circular groove. It may happen that the healthy distal part of the tooth may be entirely separated from the root by this circular caries.

If the caries attacks a tooth before its eruption is complete and then grows on, it is clear that the carious ring may not be at the neck but in

some place near the middle of the tooth, which indicates the earlier location of the gums. The earlier the caries begins the smaller is the healthy part of the tooth. The distal end which is almost separated may break off and leaves a very pointed stump.

As a rule, the upper central incisors are attacked first, then the lateral and the molars, occasionally also the canines (Meyer), while the lower teeth generally are spared. The green coating of the tooth is not a deposit, but is incorporated in the enamel and can only be removed with the latter.

The statistical collection of Neumann indicates that scrophulo-tuberculous children show this affection chiefly and Meyer (in Heubner's Polyclinic) has corroborated this discovery.

The process is not a specific expression of tuberculosis, but rather a consequence of the associated disturbance of nutrition.

The onset of the caries is attributed to an acid fermentation of the mucus in the mouth (Neumann).

VII. DISEASES OF THE SALIVARY GLANDS

The congenital anomalies and other pathological processes of the salivary glands, on account of their rare occurrence, do not excite the practical interest which is justly directed toward the inflammatory processes of the parotid in childhood.

Without considering the defects and abnormal position of single salivary glands, as well as the rarely observed cases of congenital salivary fistulæ, a few words may be given to the cysts of the salivary ducts, which are produced by a congenital atresia of the principal duct. Such abnormalities have been observed in Wharton's duct and also in the secretory ducts of the Blandin-Nuhn glands. These congenital cysts may naturally hinder the act of nursing and thus create the necessity of an operation.

A special consideration is merited by the cystic neoplasm called *ranula*. The term ranula originally was applied to all cystic growths under the tongue; in most cases it arises from the sublingual gland. It must therefore be considered to be a congenital or acquired retention cyst of this gland.

The swelling lies under the tongue, most commonly at both sides of the frenulum, which causes a depression in the centre of the growth. As a result of its position, on growing larger it forces the tongue upward. The growth is given this name on account of its shining, translucent appearance and its grayish red discoloration. The cyst is filled with a tough and sticky content which is usually colorless, but occasionally is tinted yellowish green to brown.

The presence of the growth may occasionally hinder nursing or even respiration. Its treatment is surgical.

Hennig, Mikulicz, and Kümmel described an acute primary inflammation of the salivary glands in infants, as *sialo-adenitis* of infants. It is remarkable that it never attacks the parotid but only the submaxillary and the sublingual glands. These glands become acutely swollen and high fever appears, then pus is discharged from the ducts. The disease ends as a rule with a wide-spread abscess formation, but which has a good prognosis on account of its location. Hennig favors the view that the real affection has a connection with puerperal diseases.

While the *primary idiopathic parotitis*, which is identical with the parotitis epidemica, is described at another place (see Vol. II), it is expedient here to examine more closely the inflammatory changes of the parotid which occur in the course of other diseases.

The *secondary parotitis*, like the primary form has always an infectious origin; the infectious agent reaches the gland directly through Steno's duct or indirectly by way of the circulation in the gland (metastatic parotitis). It is clear, therefore, why inflammatory processes in the neighborhood of the openings of the salivary ducts, the various forms of stomatitis, are the most frequent excitants of a secondary parotitis. Likewise otitis media is not an infrequent cause of inflammation of the parotid. The pus burrows by the way of the Glaserian fissure into the gland (Gruber).

The local phenomena, which in general resemble those of parotitis epidemica, are distinguished from this chiefly in that the secondary form usually occurs on one side only, and the swelling of the gland subsides in a few days spontaneously or very rarely suppuration of the gland takes place.

The abscess formation is not easily foretold, as palpation may reveal no fluctuation even when pus is present, since the parotid is covered by a rigid fascia. It is necessary, then, to place a greater significance on the elevation of the temperature and the augmentation of the distress than on the local findings.

The primary expectant treatment (see Epidemic Parotitis) must give place to surgical interference when the appearance of pus is evident.

Those forms of secondary parotitis which arise during the course of the severe infectious diseases,—especially typhoid and the acute exanthemata—possess a more serious character. With the increase of the glandular swelling the skin over it takes on an inflammatory redness. The course of this inflammatory process is often protracted and is a grave complication to the child already weakened by the febrile disease. Moreover, this inflammation shows a very slight tendency to subside and generally ends in suppuration or occasionally in gangrenous disintegration of the gland.

Microscopical examination has shown that this form of parotitis also is induced by an infection through the salivary duct. The name

metastatic parotitis should, consequently, be reserved for those cases which arise in the course of a septicæmia, or possibly a septicæmic typhoid, by a specific hæmatogenous infection of the gland.

A rational care of the mouth during the infectious diseases is the most effective prophylactic measure.

At the onset of the inflammation one may try an embrocation of iod-vasogen in order to hasten the absorptive process within the gland; as soon as the presence of pus is manifested a free incision must be made.

DISEASES OF THE TONSILS, PHARYNX, AND ŒSOPHAGUS

BY
DR. H. FINKELSTEIN, OF BERLIN

TRANSLATED BY
DR. CLEMENT F. THEISEN, ALBANY, N. Y

DISEASES OF THE TONSILS AND PHARYNX

ANATOMICAL AND PHYSIOLOGICAL REMARKS

THE posterior nares and fauces are surrounded by Waldeyer's lymphatic ring, a broad area of lymphoid tissue, which, at certain points develops into large tumor-like formations, designated as the pharyngeal tonsil, faucial tonsils and lingual tonsil. The lymph-channels of this region run to the cervical glands situated around the jugular vein. The drainage of a considerable portion of the lymph-vessels from the nasopharynx is to the lateral pharyngeal glands, situated behind the tonsils, in the buccopharyngeal fascia, which are intimately connected by numerous anastomoses with the deep glands of the neck. A certain number of these vessels pass the small retropharyngeal glands. The remainder of the lymph-branches, run, by way of the lateral pharyngeal glands, behind the great vessels of the neck, directly to the deep cervical glands. The lymph-channels of the pharynx however, particularly the faucial tonsils, have nothing to do with the lateral pharyngeal glands. They are distributed to the submaxillary glands, particularly those at the angle of the jaw, and from there to the superficial or deep cervical glands. All this is of diagnostic importance, because swelling of the pharyngeal and retropharyngeal glands, which are only palpable from within, points to disease of the nasopharynx; swelling of the submaxillary glands to disease of the tonsils; while swelling of the cervical glands without involvement of the submaxillary glands, can only point to some nasopharyngeal condition.

The lingual tonsil really only develops after the age of puberty, and occasionally has pathological significance. On the other hand the development of the other tonsils is often very rapid in childhood, and hyperplasia and diseases of the tonsils are so common that they make up a characteristic part of the pathology of childhood. The faucial tonsils, however, until about the second year, possess a well-known immunity, and only later on, when they become much enlarged, do they have the same pathological importance that is peculiar to the lymphoid tissue of the

nasopharynx from earliest infancy. Pathological changes of this ring of lymphoid tissue extend during childhood to all its parts with the exception of the lingual tonsil. If pathological conditions of the faucial tonsils and the pharyngeal tonsil are treated as separate conditions, recurrences will take place. It is best to consider such disturbances conjointly.

ACUTE INFLAMMATORY PROCESSES

1. CATARRHAL AND EXUDATIVE FORMS

Angina, the acute disease of the lymphatic ring, is a part of a whole list of general infections. It represents the main lesion in scarlet fever and diphtheria, and it accompanies as a more or less important disturbance the other acute exanthemata, and influenza, pneumonia, typhoid fever, etc. Angina must be considered of particular importance as being the portal of entry for articular rheumatism, and, as late investigations show, of epidemic cerebrospinal meningitis. Only idiopathic anginas will be considered.

Predisposition.—It is a well-known fact that many children are predisposed to "sore throat." Unimportant causes such as a "cold," or a voice strain are sometimes sufficient to cause a disturbance in the pharynx. Or for many years the child will have recurring attacks of tonsillitis, which may come on so frequently that a serious condition is produced. This hereditary and family predisposition often does not disappear until after the age of puberty. It is often attributed to chronic throat conditions, which lead to acute exacerbations. It is claimed that infectious material may linger in the folds and crypts on the surface of the tonsil, particularly in the form of tonsillar concretions, made up of mucus, detritus and bacteria, which, every time conditions are favorable, is started into fresh activity.

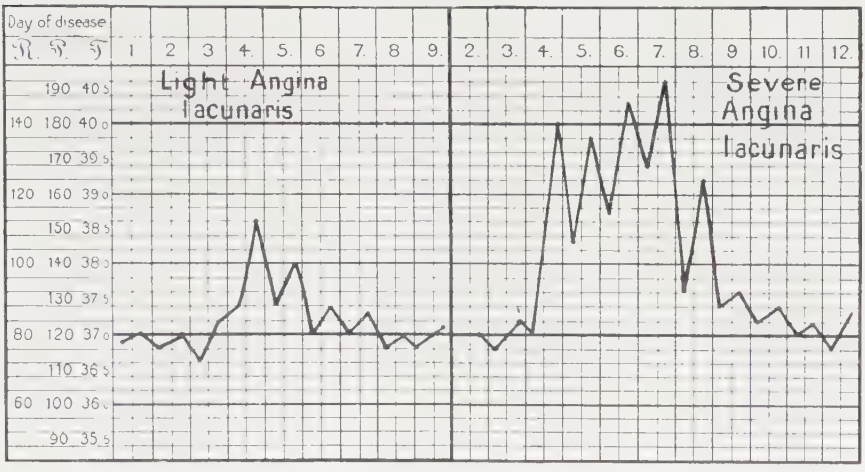
Against this view of a localized predisposition, the argument may be advanced, that children with absolutely similar conditions of the pharynx, so far as their predisposition to attacks of angina is concerned, differ very materially, nor can any positive relation be traced between such attacks and the hyperplastic condition of the tonsils, because many children with large tonsils remain entirely free from attacks of angina, while many with only slightly enlarged or practically normal tonsils, are subject to repeated attacks. Much more weight must be attached to the theory of a general predisposition. We are forced to this conclusion, because diatheses, described as lymphatic, scrofulous and more recently as exudative, with or without hyperplasia of the lymphatic apparatus make themselves apparent by an increasing susceptibility to bacterial invasion.

Etiology.—Many of the anginas of predisposed individuals are caused by auto-infection. There are however many cases which must be attributed to infection from without, attacking predisposed as well

as apparently immune individuals, which, in their way of extension, are entirely similar to the infectious diseases of childhood. It may be said, that certain cases occur in epidemics in families and institutions. In such epidemics the disease must be transmitted from one person to the other, and there is probably a specific angina poison causing such cases which persists in certain regions like a miasm. In many institutions, hospitals and orphan asylums, nearly every new inmate as well as new internes in the hospitals, have an attack of this form of angina.

The streptococcus is the organism that usually causes the disease. Other organisms such as the staphylococcus, pneumococcus, the coccus conglomeratus (Stooss), micrococcus catarrhalis (Pfeiffer), etc., are also sometimes responsible for the infection. Mixed infections must also be considered. An etiological classification of the anginas according to

Fig. 12.



Day of disease. Mild lacunar angina. Severe lacunar angina.

the bacterial findings is not practicable. Generally speaking the streptococcal diseases are the most severe, while the anginas caused by the staphylococcus and especially the pneumococcus are milder. General and local conditions may remain hidden. The clinical picture is the only reliable guide. According to this we must distinguish between catarrhal angina, follicular angina and lacunar angina. These forms have so much in common in the symptomatology, that it is best to first consider them as a whole.

General Symptoms.—Tonsillitis (angina) begins acutely, either as a general disturbance or as a symptom of disease of the upper air-passages or the digestive tract. The early symptoms are usually constitutional, because children do not always complain of even severe local lesions. The invasion is usually like that of an infectious disease. Young children look badly and are often irritable, while older children complain of lassitude, headache and chills. The temperature rises,

according to the severity of the attack to 38° C. (100° F.) and even 40° C. (104° F.). Gastro-intestinal disturbances, vomiting and diarrhœa are quite common. Young children sometimes have convulsions. Older children (although not always) complain of difficulty in swallowing.

The presence of freshly swollen, slightly painful submaxillary glands and an odor from the mouth which may be absent in children without teeth are important symptoms. A considerable swelling of the mucous membrane of the throat may be recognized by the thick speech, and some difficulty in breathing which may give rise to a distinct stridor. Inspection will show characteristic findings in the different forms.

Catarrhal Angina

In catarrhal angina the pharyngeal mucosa is much reddened, more or less swollen and coated with mucus; the tonsils are sometimes decidedly swollen, at other times only slightly. Small hæmorrhages are sometimes visible in the epithelial layer. There are sometimes circumscribed inflammatory areas, at other times the membrane is generally inflamed. Catarrhal angina includes most of the milder forms of sore throat although there are cases with severe disturbances. It usually lasts from two to three days, rarely a week.

Follicular Angina (*see Plate* 37)

Nomenclature.—Follicular angina and lacunar angina are used by many authors as synonymous terms while others distinguish two distinct conditions. Under follicular angina should be included cases in which the lymph-follicles of the tonsils and other adenoid tissue are inflamed and swollen. In lacunar angina there is an inflammatory exudative process of the surface of the tonsil, localized mainly in the crypts and the tissue surrounding them. Follicular angina is differentiated from the catarrhal form by the swollen lymph-follicles under the mucous membrane, which at first appear as grayish yellow dots, later studding the surface of the tonsil in the form of yellowish elevated points. The swelling of the follicles either rapidly subsides, or they rupture, producing a small superficial ulcer which heals rapidly. The rounded spots, and the absence of confluence, differentiate this condition from lacunar angina.

Lacunar Angina (*see Plate* 37)

Angina lacunaris is characterized by the development of a grayish yellow, or yellow, mucopurulent exudate, on the surface of the tonsil, in the beginning often covering the whole tonsil, but in a short time taking on a distinctly lacunar type. The tonsil is swollen, the rest of the pharynx presenting the picture of a catarrhal inflammatory process. It may be unilateral or bilateral. Constitutional symptoms, glandular swelling,

PLATE 37.

I

II

III

IV

I. Angina lacunaris.
II. Angina follicularis.
III. Hyperplasia of tonsils.
IV. Angina lacunaris.

and difficulty in swallowing, are more severe than in the forms just described. It usually runs its course in two or three days, but may last a week or longer; a longer course is produced by first one side and then the other becoming involved. The exudate is thrown off on the second or third day, leaving superficial epithelial erosions, the other symptoms disappearing soon after.

Retronasal Angina

(Pharyngitis superior, pharyngeal angina, adenoiditis, amygdalite pharyngée)

Retronasal angina was formerly not considered a distinct condition, and its importance has only been recognized during the last few years. The recognition of the condition is of great importance during childhood. Its clinical picture is very much like that of angina of the tonsils. One of the first symptoms is the difficulty in breathing, with the nasal voice, and some earache and deafness. There is a profuse mucopurulent discharge from the nose, and tenacious mucus will be seen clinging to the posterior pharyngeal wall, which is reddened and presents an uneven surface owing to the swollen patches of lymphoid tissue. If posterior rhinoscopy is possible, it will be seen that the pharyngeal tonsil is also subject to catarrhal, follicular, and exudative inflammatory processes. If an examination is made with the finger the swelling in the vault of the pharynx can be felt. The cervical glands are swollen. Its course is very much like that of lacunar angina, long-continued elevations in temperature being not uncommon however.

Unusual Courses.—These conditions do not always run a typical course. Gastric symptoms, with continued fever and an enlarged spleen, show a picture very much like typhoid. They also occasionally simulate meningitis. In small children with irritable nervous systems, the acute febrile affection may cause nervous symptoms. The fever, particularly in retronasal angina, sometimes runs a very unusual course, being at times intermittent at other times remittent, and accompanied by chills. Some very susceptible children have attacks of this kind at such short intervals that a chronic form (Fischl) is produced, causing a severe general disturbance.

Complications.—These occur mainly in the retronasal and lacunar varieties.

(*a*) *Parenchymatous tonsillitis, tonsillar and peritonsillar abscesses.*—Occasionally a severe parenchymatous swelling will develop in either one or both tonsils, and at times in the pharyngeal tonsil. All the throat symptoms become much aggravated, and there is a characteristic stiffness of the jaws and head. The tonsils are intensely inflamed and swollen, with pain, and a high remitting fever. Inability to take nourishment soon reduces the patient very much. Some of the cases abort, while others go on to the formation of a peritonsillar abscess. After

evacuation of the abscess, convalesence is rapidly established. The pus usually contains streptococci. This complication is more frequent in adults than in children.

(*b*) *Lymphadenitis.*—The regional glands at times remain much swollen, causing prolonged elevations in temperature. The submaxillary and cervical group of glands are involved, at times a single gland or group of glands, at other times several on one or both sides. In small children, the inflammatory process often jumps from one chain of glands to another. Under the name "glandular fever," Filatow and E. Pfeiffer have described a condition accompanied by fever, constitutional symptoms, an inflamed pharynx and acute swelling of the upper posterior cervical glands, running a course similar to lymphadenitis. Pain between the sternum and navel is sometimes present, and is attributed by Pfeiffer to an involvement of the mediastinal and mesenteric glands. Nephritis is an occasional complication. This condition cannot be considered a distinct entity. It is probably the result of a primary retronasal angina with slight local symptoms, but with a severe involvement of the regional glands (Hochsinger Zappert, Trautmann).

(*c*) Severe forms of *otitis media* are frequent complications of retronasal angina, and it is in rare instances the starting point of a *meningitis.*

(*d*) *Erythema* and *exanthems* simulating scarlet fever are not uncommon.

(*e*) Catarrhal affections of the larynx, bronchial tubes and lungs also follow this variety of angina.

Secondary Diseases.—In a certain percentage of angina cases, diseases of other organs follow, particularly "rheumatic" conditions: erythema nodosum and exudativum, purpura, swelling of the muscles, inflammation of the joints, inflammation of the endocardium and other serous membranes and hæmorrhagic nephritis. In particularly unfortunate cases, the septic poison is taken into the system, causing septic or pyæmic metastases. Attention has also been called to the combination of angina and appendicitis.

Diagnosis.—The onset of an angina may simulate typhoid fever, pneumonia, meningitis, or an acute digestive disorder. If a routine examination of the throat is practiced in all diseases of children, mistakes of this kind in diagnosis will not be made. It is important to examine for swelling of the regional glands. From scarlet fever, a differential diagnosis may be made by the absence of the eruption and the course of the fever and the more diffuse redness of the pharynx. The redness of the throat is less sharply defined in scarlet fever. Cases of angina with an accompanying erythema may be very confusing.

Scarlet fever may also occur without the eruption. Cases of lacunar angina are very common during epidemics of scarlet fever. The character of the exudate is different in lacunar angina from that in diphtheria,

and it is confined to the tonsils. There are however, cases of diphtheria that simulate lacunar angina so closely that only a bacteriological examination will clear up the diagnosis.

Prophylaxis.—As so many anginas are infectious, it is advisable to isolate every case.

Treatment.—In uncomplicated cases rest in bed, liquid diet, with proper applications to the neck (warm or ice in severe cases), when possible, gargles of chamomile tea, salt water and glycerin, water and lemon juice, or peroxide of hydrogen, one-half teaspoonful to one-half glass of water, will be effective. Mercurial preparations may be given internally but are really unnecessary. In young children a 2 per cent. solution of potassium chlorate may be given internally. The much advertised angina lozenges give very little relief.

Local interference is only indicated for abscess formation. An early incision will shorten the attack materially. Fluctuation may be felt with the finger.

The incision when an abscess has formed is made in about the centre of the anterior faucial pillar, slightly beyond its border. Warm gargles may be used after the abscess is opened. The removal of the tonsils, or incising the crypts for the purpose of preventing recurring attacks, are of doubtful value. A chronic or recurring inflammation of the lymphoid tissue in the nasopharynx, with excessive nasal secretion, may be practically cured by curettage of the nasopharynx and long-continued elevations in temperature checked. The removal of the faucial tonsils is not followed by such good results. If the pharyngeal tonsil is removed at the same time, the results might be better.

Much can be accomplished in the way of overcoming such susceptibility to attacks, by diet and climate. The so-called "hardening" process is useless.

2. ULCEROMEMBRANOUS ANGINA

(Angina ulcerosa, Plaut's and Vincent's angina. Angina bacille fusiforme, diphtheroid angina)

This disease was first described clinically by Russians (Szimanowsky and Filatow), and by French authors (Barthez and Sanné). The first discoveries of etiological importance were made by Plaut in 1894, who first called attention to the fusiform bacillus found in such cases. Vincent published his observations in 1898.

Symptoms.—The disease has an acute onset, but unlike other forms of angina, the constitutional symptoms are slight. This slight general disturbance with severe local lesions is characteristic. The inflamed and swollen pharyngeal mucous membrane has a tendency to bleed. Sharply defined ulcers, covered with a tenacious secretion, then develop, usually on one tonsil.

Two forms are described: a diphtheroid, in which a pseudomembrane, covering a slightly eroded surface, develops, not resulting in much destruction of tissue; and an ulceromembranous form, in which deep ulcers appear on the tonsils.

Course.—The first variety runs a mild and rapid course, healing like an ordinary angina. In the second variety, the ulceromembranous form, healing is much slower. The membranous coating is not thrown off before the end of the first week or even longer, and the ulcers heal slowly. The majority of the patients get well, but in exceptional cases extensive necroses with a fatal outcome have been observed (Bruce, Ellermann, Mayer and Schreyer).

Etiology.—The infectious nature of the disease has been well established, as many house and family epidemics have been observed. Some authors (Bernheim and Pospischill), regard the disease as an atypical localization of an ulcerative stomatitis. Inoculation experiments on healthy persons have not succeeded (Uffenheimer). Certain bacteria and spirochæte appear in great numbers sometimes in pure culture even on cover-slip preparations. The fusiform bacillus, not at all, or slightly, movable, is colored with difficulty by Gram's method. A long, thin, movable (negative to Gram) spirochæta occurs in conjunction with this. Ellermann, after a long trial, has succeeded in obtaining the fusiform bacillus in pure culture as a strict anaërobe. The etiologic rôle played by these symbiotic bacteria is shown by their constant presence, and by the fact that they have been obtained in pure culture from the deepest layers of the mucous membrane (Hess, Gross, Ellermann, Graupner, Beitzke).

The same, or at least similar, organisms have been found in other ulcerative processes, such as noma. They have also been found in healthy mouths, in carious teeth, gonorrhœal stomatitis, and chancre of the tonsil (Jürgens, Hahn). These findings however should not be considered as counting against the etiological importance, because in pathology a great many instances are found in which the same organisms, which as saprophytes exist harmlessly on body surfaces, under favorable conditions may develop pathogenic properties. The spirochæte appear to be important factors in the production of the bad odor from the mouth.

Diagnosis.—This is made by the appearance of the pharynx, the bad odor, and the bacteriological examination. It must be differentiated from diphtheria and syphilis. This is often difficult, because diphtheria bacilli and angina microbes frequently occur together. In the same way angina and syphilitic ulceration occur simultaneously. The diagnosis must be made by exclusion. An examination for diphtheria bacilli should always be made.

Treatment.—This is similar to that for angina. Potassium chlorate has been recommended. The removal of the tonsil to shorten the attack

has been suggested, but it has not been determined what the result would be (Uffenheimer).

3. GANGRENOUS ANGINA

Cases of primary severe gangrenous inflammation of the pharynx have appeared in the literature, and may be distinguished from the severe cases of ulceromembranous angina by the absence of specific bacterial findings and the presence of a particularly frightful odor. Many cases die with the symptoms of a severe septicæmia. A hæmorrhagic diathesis is common. The prognosis is doubtful. Maurin saw four out of five cases of circumscribed gangrene recover, and only two out of five in which the gangrenous process was more extensive.

The cause of this affection is not known, but it bears no relation to diphtheria.

4. SEPTIC PHLEGMON AND ERYSIPELAS OF THE PHARYNX

Pseudodiphtheritic Pharyngeal Necrosis

Seropurulent and erysipelatous diseases of the pharynx, with severe general septic symptoms, occur in nursing infants. The process is similar to that in adults. The occurrence of pseudodiphtheritic necrosis of the pharynx is confined to poorly nourished children in the first few weeks of life. The destruction of tissue may extend to the bone. The destructive process extends from the pharynx to the nose, and epiglottis, and may extend through the œsophagus to the stomach, while the larynx and trachea remain exempt. The affection always ends in death, with symptoms of a general septicæmia. It is probable that the affection is caused by inoculation with septic bacteria, during the process of wiping out the mouths of poorly nourished children.

HYPERPLASIA OF THE TONSILS

While hyperplasia of the faucial tonsils has long been a recognized condition, it is only since 1868 that the same anomally of the pharyngeal tonsil has received attention. Wilhelm Meyer of Copenhagen, was the first to call attention to this condition which plays such an important rôle in the pathology of childhood.

Anatomy.—The hyperplasia as a rule involves all three tonsils equally. This enlargement of the tonsils either takes the form of a compact tumor-like, or polypoid, pedunculated swelling. The growth may be firm or soft. In the tonsillar crypts concretions made up of detritus and bacteria are frequently found. Microscopically, the structure of the soft tonsil is found to be simply lymphoid tissue, while in the firm tonsils there is a formation of connective tissue showing a previous chronic inflammation.

Causes.—The causes leading to tonsillar hyperplasia have not been definitely determined. One view is that the enlarged tonsil is the result

of many attacks of acute inflammation; on the other hand it is believed that primary hyperplasia occurs, this tissue being particularly susceptible to inflammatory processes. This view is undoubtedly the correct one. These hyperplasias occur independently of catarrhal or recurring inflammatory processes, and are probably due to a distinct predisposition, known as the "lymphatic constitution," on the part of the individual.

Tuberculous or scrofulous diatheses are not factors in such hyperplasias. Neither need they be necessarily regarded as a result of constitutional weakness leading to catarrhal conditions of mucous surfaces (the exudative diathesis of Czerny).

Fig. 13.

Facial expression in adenoids.

There are many such susceptible children free from enlarged tonsils, and on the other hand many children with enlarged tonsils who are not susceptible to catarrhal conditions.

It is better to consider tonsillar hyperplasia as the expression of an increased tendency to the formation of lymphoid tissue during childhood.

1. HYPERPLASIA OF THE PHARNYGEAL TONSIL

(Adenoids, adenoid vegetations)

Frequency.—Adenoid vegetations are extremely common during childhood. Kafemann found that 17 per cent. of school children examined by him had adenoids, Schmuckmann 30 per cent., Felix from 28.52 per cent. to 35.1 per cent., and Wilbert found that 62 per cent. of children had adenoids. The condition is most common between the

sixth and eleventh years, and fairly uncommon after the age of puberty. Körner has found adenoids in 36 per cent. of sea-coast dwellers.

Symptoms.—The symptoms depend largely upon the size of the growth in the nasopharynx, and on the presence or absence of inflammatory complications.

(*a*) *Symptoms Caused by Nasal Obstruction.*—The nasal obstruction results in mouth breathing, and this is responsible for the characteristic facial expression of children with adenoids. The mouth is kept open, the lips are dry, the nasolabial folds are drawn down, and the eyes are dull and heavy, giving the face a stupid expression. Many of the children are in poor physical and mental condition. This deficient mentality is caused by impairment of hearing, more than two-thirds of the children with adenoids being deaf (Abeles, Halbeis, Hartmann, Wilbert). This deafness is the result of obstruction of the pharyngeal ostium of the Eustachian tube, leading to deficient ventilation of the middle ear, salpingitis, and retraction of the tympanic membrane. The voice lacks resonance, and children often snore during sleep. Headache is quite common. Deformities of the bones, particularly of the superior maxillary, consisting in a highly arched hard palate and pointed alveolar process, also result from the interference with nasal breathing. Irregularities of the teeth are also common. Faulty development of the thorax (flattening, chicken breast, Tröltsch, Haug, Hopmann), probably occur only in cases of rachitis, and the nasal obstruction can only be considered one of many etiological factors. Spinal curvatures have also been observed (Redard, Ziem).

FIG. 14.

Exophthalmos with adenoids in boy aged seven years.

Exophthalmos is an interesting complication (Holz, Spieler). Instead of attributing this to a function of the hyperplastic tonsil similar to the thyroid (Holz), it is probable that a retrobulbar lymph-stasis (Spieler), or anomalies of the orbit (Escherich), play the main rôle.

(*b*) *Inflammatory Complications.*—Swelling of the cervical glands shows how intimately adenoids and inflammatory processes are associated, and such involvement of the glands is of great symptomatic and diagnostic importance. The faulty nasal breathing explains the frequent occurrence of catarrhal conditions, which may be mild or severe. They are not limited to the tonsils, but may involve the nose as well as the pharynx (follicular pharyngitis). A stubborn mucopurulent rhinitis is a common symptom in children, and often leads

to hyperplastic conditions of the nasal mucosa. Complications on the part of the middle ear and respiratory passages are common.

(*c*) *Effects upon Remote Organs and their Functions.*—Catarrhal conditions of the larynx, and collections of tenacious secretion in the pharynx produce severe spasmodic coughs with retching, vomiting and even spasm of the glottis. Night terrors are not uncommon. There is no positive proof that asthma, chorea, epilepsy, enuresis and other neuroses are the result of adenoids, but speech disturbances, particularly stam-

FIG. 15.

Exophthalmos with adenoid vegetations (nursing infant).

mering, may have some connection with this condition. Adenoids occur so frequently also in stuttering (Kafemann, Berkhan, Karutz, Pluder), that it is wise to begin treatment of such cases with an adenotomy.

Importance of Adenoids to the General Health.—Physical and mental deficiencies are common symptoms. Children are unable to concentrate the attention, they are indifferent, and do not keep up with children of their own age (nasal aprosexia, Guye). It is of the greatest importance to recognize the symptoms on the part of the ear. Deafness and various other aural diseases are common.

Diagnosis.—The symptoms already described, *i.e.*, the appearance of the child, the speech, deafness, and the mucopurulent secretion on the posterior pharyngeal wall make the diagnosis easy. Hypertrophic rhinitis, a congenital smallness of the nasopharynx, or other tumor, may however lead to errors in diagnosis. A direct inspection of the nasopharynx by posterior rhinoscopy, or examination with the index finger of the right hand, will clear up all doubts.

Treatment.—An operation (adenotomy) is the only form of treatment for most cases. This should be performed, even in nursing infants, as soon as its necessity has been determined. Hæmophilia, and acute inflammatory processes in the pharynx, would be contraindications. Medical treatment and local applications will not do much good. The operation should however only be performed when positive symptoms, directly caused by the adenoid growth, are present, as for example, mouth breathing, nasal speech, and deafness. A decision is more difficult when inflammatory complications occur. The operation is of doubtful service when there is only a small amount of lymphoid tissue causing recurring catarrhal attacks. Such attacks often go with general constitutional disturbances. A chronic purulent rhinitis, pharyngitis, or an otitis media, are usually taken to be positive indications for an operation. Enlarged glands in the neck occasionally show improvement after adenotomy.

Fig. 16.

Pointed cranium with adenoid vegetations.

To perform the operation, curettes (Gottstein's, Beckmann's, Kirstein's and Fein's) are in common use. Juraz and Schech's forceps and the adenotome are also used a good deal. The child is seated on the lap of an assistant and held firmly, the mouth being held open with a tongue depressor or preferably a mouth gag (Beckmann's or Jansen-Windler's). The curette is then inserted back of the velum between the growth and the posterior nares, carried to the vault of the pharynx and swept downwards, the growth being often carried out of the mouth with it. The piece often drops into the throat and is swallowed. Fragments left behind may be removed with forceps. The bleeding which is at first severe soon ceases. General anæsthesia is recommended by some phy-

sicians, and is not employed at all by others. It is well to examine with the finger to determine whether fragments are left behind.

Deep anæsthesia at any rate is unnecessary. The child should be kept in bed for a short time and on a soft diet. Secondary hæmorrhages are rare, and occur either at once or one or two hours after the operation. They are produced either by hæmophilia, or by the presence of only partially removed adenoid fragments. If the usual hæmostatics, (adrenalin) fail, tampons or gelatin may have to be employed. Fatal cases are recorded. Secondary fever occurs at times, and is usually due to retained pieces of the growth, which lead to inflammatory symptoms. Graver complications such as post-operative paratonsillar or retropharyngeal abscesses occasionally occur. Severe septic conditions of the mouth and pharynx have also been reported. Careful asepsis should be observed in operating.

The results of the operation are excellent. Recurrences occur in a small percentage of cases operated upon, but as a rule are not very common if a thorough operation has been performed. They cannot be positively prevented however.

2. HYPERPLASIA OF THE FAUCIAL TONSILS (see Plate 37).

Occurrence.—Enlarged faucial tonsils are rare in nursing infants. They develop usually about the second year, but even at this age may be so large that considerable trouble may be produced.

Symptoms.—Enlarged tonsils may be easily recognized when the throat is inspected, as they project as either round or oval shaped tumors. They often cause the faucial pillars to be widely separated. Deposits in the crypts are frequent. Slight enlargements of the tonsils may not produce any symptoms, but when larger and associated with adenoids they always do. The voice has a muffled sound, and where the pharynx is much filled up, a pharyngeal stridor is present.

Diagnosis.—The diagnosis is made by examining the throat.

Treatment.—A moderate enlargement causing no symptoms will not require treatment. When the tonsils are subject to recurring inflammatory attacks, they should be removed. The galvano-cautery snare is often used for this purpose, although the ordinary tonsillotomes (Mackenzie, Babinsky, Matthew, Fahnenstock), which encircle the tonsil with a ring knife, are handier. The lower portion of the tonsil should also be removed. Hæmorrhage is usually slight although secondary hæmorrhages occur. If the ordinary styptics, adrenalin, gelatin, hot water, fail, pressure with the finger, or with the compressor of Mikulics or Springer, will control the bleeding.

A membrane forms over this cut surface which may be mistaken for diphtheria. It must be borne in mind however, that true diphtheria may occur in the wound.

Growths of the Tonsils and Pharynx

Benign and malignant tumors may occur in the nasopharynx during childhood. Dermoid cysts, lipomata, fibromata and fibrosarcomata have been reported. Lymphosarcoma of the tonsil has also been observed.

Retropharyngeal Lymphadenitis. Retropharyngeal Abscess

Etiology.—Inflammatory processes leading to abscess formation in the retropharyngeal and lateral pharyngeal glands may occur. This may be brought about by infection from ulcers or from a "diffuse infectious catarrh?". Syphilis, measles, scarlet fever, rhinitis, and retronasal angina are important etiological factors.

A true idiopathic retropharyngeal lymphadenitis does not exist.

Streptococci are almost always found on bacteriological examination, but influenza bacilli and other bacteria are also found.

Occurrence.—It usually occurs during the first year of life. Out of Bókay's 467 cases, 296 occurred in the first year, and 78 in the second. It is believed that later in life these glands become obliterated. Like catarrhal conditions, this disease occurs in winter and spring.

Pathological Anatomy.—At first there is a simple swelling of the gland, but later on a periglandular infiltration develops. Abscess formation is common.

Symptoms.—Difficulty in swallowing is one of the first symptoms. There is an excessive collection of mucus above the obstruction. Hoarseness is often present, and the child breathes with open mouth and with a pharyngeal stridor. The head is held rigidly and to one side. The gland may be felt with the finger, usually laterally behind the pillars of the fauces. Later the gland which is at first movable, becomes immovable, and finally fluctuates. The tumor may at times be readily inspected. Sometimes attacks of suffocation develop. Deep seated swelling may simulate laryngeal croup.

Course.—Some cases subside gradually, others go on to abscess formation, which opens spontaneously or is incised. An involvement of the neighboring glands is common. The purulent process may extend from one gland to another, so that there will be a large collection of pus in the throat. This sometimes burrows in the region of the parotid gland or into the mediastinum.

The spontaneous opening of an abscess may cause death by a septic aspiration pneumonia. Cases of pyæmia have also occurred.

The **prognosis** is favorable when the condition is recognized early and promptly treated.

Diagnosis.—The condition is often overlooked by beginners. The pharyngeal stridor, the position of the head, the hoarseness, the ratt-

ling of mucus in the throat are typical diagnostic signs. Examination with the finger will settle all doubt.

Treatment.—The treatment of the tumor before fluctuation, is like that of any angina. A prompt incision should be made with the finger as a guide; the knife blade being covered to within a short distance of the point, or with the knife of Schmitz or Carstans. This should be done with the child in the upright position, but as soon as it is made the head should be lowered to prevent the aspiration of pus. Recovery, except in the cases in which large collections of pus have formed, is rapid. In such cases the external incision may be advisable (Schmidt, Oppenheimer, Burckhardt). Occasionally tuberculous swelling of the retropharyngeal glands, or abscess of other glands occur. Such conditions run a chronic course. Operative interference should be delayed as long as possible, and other methods of treatment, such as injections of iodoform may first be tried.

DISEASES OF THE ŒSOPHAGUS

(Corrosive Œsophagitis)

Etiology.—The unfortunate cases in which caustic chemical poisons are swallowed by children, causing burns in the œsophagus, are fairly common. The substance most commonly swallowed is lye, in the form of washing fluid.

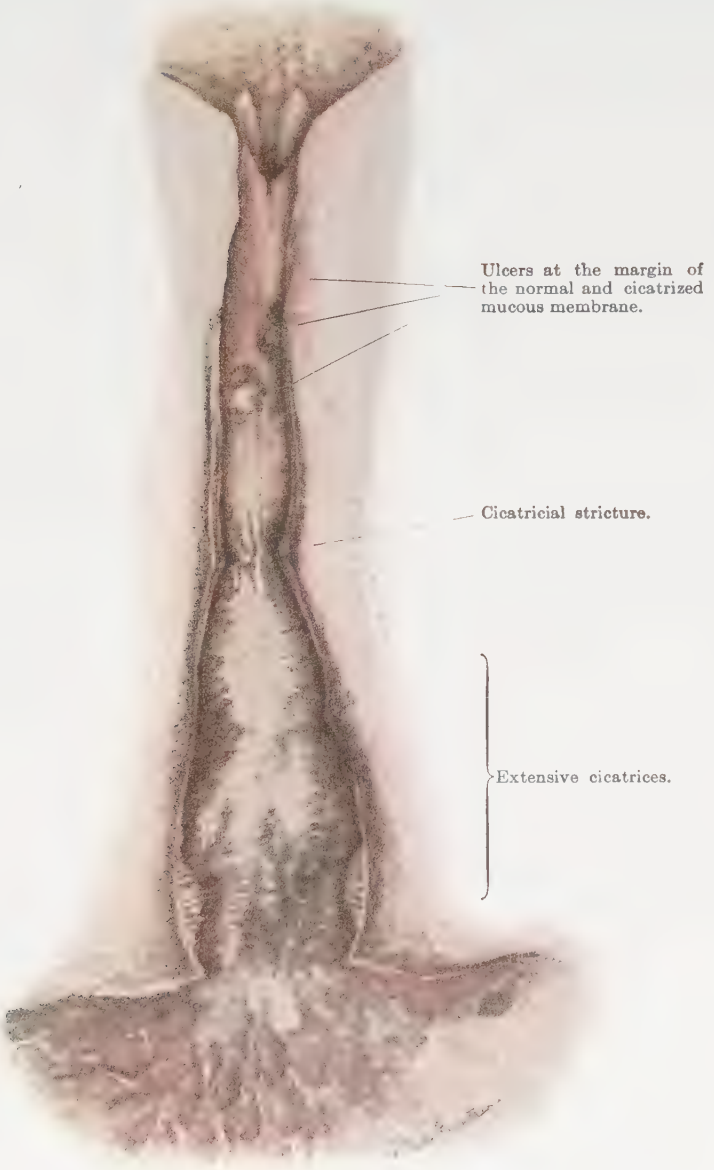
Pathological Anatomy.—Slight burns do not produce cicatrices, but only necrosis of the epithelium. Severe lesions destroy the entire thickness of the mucous membrane, sometimes even the œsophageal walls. Extensive ulceration is produced, which heals by cicatricial tissue, resulting in the formation of strictures.

Symptoms.—After the poison is swallowed, masses of bloody mucus are expelled, which in severe cases may contain portions of mucous membrane.

Deglutition becomes very painful, and the general condition is influenced by the severity of the infection. Serious collapse occasionally occurs with a fatal termination. In the other cases, recovery, which may be interrupted by other serious symptoms, such as sloughing of the tissues, erosions of the blood vessels and hæmorrhages, perforation, with phlegmon of the neck, or mediastinitis, with emphysema of the skin or pyopneumothorax, ensues. In the milder cases such complications do not occur, but after several weeks, new symptoms, caused by the formation of cicatricial tissue with stricture, develop. According to von Hacker, one quarter of the patients die as a result of swallowing lye, while sulphuric acid poisoning causes a mortality rate of fifty per cent. In more than one-half the cases, serious strictures result; of the other cases some develop slight and others no strictures at all. About one-third of the patients with stricture die.

Treatment.—Immediately after the poison is taken antidotes (chalk, magnesia, vegetable acids) should be given.

Fig. 17



Ring-like stricture (corrosive) of the œsophagus. Boy, aged 13 years.

The further treatment consists in quieting the pain and looking after the collapse which may ensue. Hypodermics of morphine according to the age of the patient may be used. Ice may be administered internally. Non-poisonous local anæsthetics like anæsthesin may be

tried. It is useful, used in an oily solution in cases of lye poisoning. Nourishment should be liquid and if necessary rectal. Instruments for dilating the stricture should not be employed for from two to four weeks after poisoning. If a sudden occlusion of the œsophagus takes place as a result of sudden swelling gastrotomy may have to be performed (v. Hacker).

CICATRICIAL STRICTURE

Etiology.—Strictures of this kind in children, with the exception of rare cases due to necrotic processes following scarlet fever, and diphtheria, are produced by corrosive injuries just described.

Pathological Anatomy.—The kind and severity of the stricture depends upon the extent of the injury. Superficial lesions produce the membranous strictures. Deeper destruction, involving the muscular layer or even the periœsophageal tissue, produces the ring- or tube-like, very firm stricture. The situation of the stricture will depend upon the amount of the caustic substance swallowed, and the way it occurred. When large swallows are taken, the fluid reaches the cardia at once and causes deep burns, but when only a few drops are taken the fluid does not go down very far. In children the lesions are usually situated in the upper third of the œsophagus. Torday saw 54 per cent. in this situation, as compared to 19 and 27 per cent. in the middle and lower third. Above the stricture there is usually dilatation with hypertrophy of the wall.

The **symptoms** are those produced by the inability to swallow, and if the stricture is tight, rapid inanition may result. They begin two or three weeks after the poison is taken.

Diagnosis.—This is made by passing solid bougies of whale bone with olive-shaped metal tips.

Prognosis.—This is better in children than in adults. With proper treatment from 54 to 66 per cent. are cured. Recurrences may take place. Tube-like strictures are the most unfavorable.

Treatment consists in the gradual dilatation with bougies. For this purpose flexible bougies, such as Phillips' uretheral bougies, answer very well. For tight strictures conical, and for wider strictures, cylindrical instruments may be used. In very tight strictures the passage may be found by the careful use of thin guides contained in a hollow bougie. A drainage tube carried down with a fine probe will be useful in some cases. It may be carried through the nose with the Bellocque cannula, and left in place some time. It may be used for feeding the patient. Gradual dilatation of the stricture results from the use of constantly larger instruments, which when the parts become tolerant may be left in place for a half hour. Great care should be exercised in using instruments. Every second or third day a little of a 15 per cent.

thiosinamin solution (alcoholic) may be used. The treatment may be started about the third week after the poisoning. If the bougies are used three times a week, the treatment usually takes about six months. In order to avoid recurrences the bougies should be passed occasionally. The dangers in using the bougies consist in producing a false passage, or perforation.

If nothing is accomplished by the gradual dilatation with bougies a gastrotomy may have to be performed.

CONGENITAL ATRESIA AND STENOSIS OF THE ŒSOPHAGUS

Congenital occlusion of the œsophagus, a rare defect, is situated slightly below the larynx or at the bifurcation. Sometimes there is a communication with the air-passages. Such children regurgitate,—with symptoms of suffocation,—the smallest amount of food. The bougie will strike an impassible barrier. Children with this condition usually die during the first two weeks. There is no treatment. Cases of congenital stricture also occur. They differ from the cicatricial strictures by the history of the case, and anatomically by the presence of normal tissue. Difficult deglutition, and a tendency to regurgitation, are the symptoms of this rare condition.

THE DISEASES OF NUTRITION IN INFANCY

BY
PROFESSOR R. FISCHL, OF PRAGUE

TRANSLATED BY
DR. CHARLES HUNTER DUNN, BOSTON, MASS.

Definition.—I choose with design the term used by Czerny and Keller in their text book, "Des Kindes Ernährung, Ernährungskrankheiten and Ernährungstherapie" (The Nutrition of Childhood, its Pathology and Treatment), for the description of this group of affections of early childhood, because the group derives from this term a sharper distinction than is usually allotted to it. I shall include under this heading exclusively those diseases which are connected with failure of nutrition in the widest sense of the term, such as are due to the unsuitable character of the food, and to its inadequate digestion, absorption, and assimilation in their various stages, with symptoms referable to the digestive organs. The entire group of cases of secondary gastroenteritis and septic vomiting and diarrhœa, which appear partly as accompanying symptoms of infectious processes localized elsewhere, and partly as the symptoms of reaction against toxic influences working upon the entire organism, will not be considered here, and will only be mentioned in speaking of the differential diagnosis.

I. EVOLUTION OF OUR KNOWLEDGE OF THE DISEASES OF NUTRITION IN EARLY CHILDHOOD

Both the clinical aspect and statistics of the mortality of the first years of life disclosed to physicians the important significance and the fatal character of the diseases, which, in many localities almost decimate the rising generation, and have from the earliest times excited interest in their etiology and aroused the therapeutic endeavors of all earnest observers.

It would lead us too far afield, would not conform to the purpose of this book, and would but slightly further our knowledge of the processes to be described here, to unroll the entire tedious evolution of our knowledge of the symptomatology, essential nature, and treatment of the acute and chronic disturbances of nutrition in early childhood. This shall only be done in so far as the observations have been of value in pointing the way, and in providing new standpoints for the understanding and treatment of the affections under discussion.

The names of Bretonneau (1818), Parrish (1826), Dewees, Billard (1830), Trousseau, Bouchut (1845), Rilliet-Barthez, Virchow and von Widerhofer (1880), are associated with the clinical aspect of the matter, and with the treatment corresponding to the etiological views held at the time. These authors, with the master-eye of gifted observers, uninfluenced by secondary considerations, so thoroughly comprehended, described, and created a clear conception of the symptoms of the various forms of gastro-enteric affections, that the characteristics established by them have for the most part survived the changes caused by our later point of view as to their etiology, and our newer methods of clinical observation. The types described by them, such as "cholera infantum," "enteritis follicularis," "atrophy," and in a certain sense the "athrepsia" described by Parrot (1877) have so thoroughly penetrated our medical consciousness, that all the progress of knowledge in this field has not been sufficient to eradicate them.

The recognition of the seasonal relation of gastro-enteric cases, to the occurrence of high summer temperature, as well as to a certain level of surface water, was brought out chiefly by American authors, from whom we derive the term "summer complaint," which term has been adopted by numerous German, French, and English writers. The etiologic basis of these summer diarrhœas has been attributed to contaminated milk. Such cases have also been considered analogous to the heat-strokes of adults. All these observations have been significant of further progress in the working out of the etiology of the subject.

The decade of bacteriologic discovery from 1880–1890 which we have to thank for much new light on the question of causation of disease, did not neglect this particular division of human pathology. The important researches on the normal intestinal flora of newborn and nursing infants, undertaken and carried on by Escherich, ushered in the work, and brought us near enough to attack the pathological conditions from seemingly solid ground. The following years produced such researches as those of Escherich himself, Lesage, Booker, Baginsky, and many others. The literature on this subject, of which A. B. Marfan has compiled an excellent résumé in his monograph "Les Gastroentérites des Nourissons," (The Gastro-enteritis of Infancy) 1900, extends immeasurably, till it reaches a provisional conclusion which is essentially widely different from the original starting point. Especially noteworthy are the interesting researches of H. Tissier (1900) on the normal and pathologic intestinal flora; those of Nobécourt (1899–1904) on the significance of the association of different organisms in the pathogenesis of intestinal diseases; the works of Escherich and his pupils, Spiegelberg, Hirsh, Libman, Moro, and others, on specific intestinal infections in infants (colicolitis and streptococcus enteritis); and finally the published investigations of American physicians col-

lected by Flexner and Holt, on the significance of the various types of dysentery bacilli in the pathogenesis of infantile intestinal infections.

Contemporaneously with and partly preceding the bacteriologic era came the work of von Ritter, Klebs, Epstein, Czerny-Moser, R. Fischl, Hutinel, with their pupils, with whose work is associated on the ground of further hospital observations that of Finkelstein, Heubner, Escherich, Blum and others. These writers investigated the special character and peculiar etiology of gastro-enteric cases occurring in foundling homes and infant hospitals, and demonstrated the widely varying course of such cases from that of summer diarrhœa, the frequent absence of injuries to digestion as causes of disease, and the occurrence of epidemics and infection by contact. The septic character of some of these cases has been mentioned, and I have proposed the term "sepsis with gastro-intestinal symptoms" for cases in which gastro-enteric symptoms are only clinical appearances occurring in the course of an infection running an entirely different course.

Pasteur's discoveries of the bacterial uncleanliness of our means of nourishment drew attention—already aroused by the course of cases of summer diarrhœa—to this source of infection. Its significance appears clearly to result from the rapid and enormous increase in the bacterial contents of cow's milk (Miquel, Escherich-Cnopf, 1890). The ingenious discovery through Soxhlet of simple apparatus for the sterilization of milk was received with the most joyful expectations. Nevertheless this method disappointed the hopes raised, its failure being proven by the fact that in the course of twenty years infant mortality was scarcely affected. Thorough researches into the above method of sterilization show what defects are inherent in it, and what dangers it conceals (Flügge, 1894, Marfan, 1900).

The poor results of the artificial rearing of infants with nourishment sterile in the bacteriologic sense, and the frequency of chronic disturbances of nutrition in children so nourished, suggested that the cause should be sought in the differences in composition and adaptability of human and animal milk. A number of successive publications reaching to the most recent time followed. They begin with the work of Biedert, Camerer, and Pfeiffer, who pointed the way in a number of researches on the differences in various kinds of milk, and their significance in the infantile intestine. Schlossmann, Knöpfelmacher, Selter and others confirm the value of these researches, while Heubner, Salge, and Bendix consider them of no great importance. These researches cumulate in the publications of Czerny and his pupils (beginning in 1897) together with those of Bendix, Terrien, and Pfaundler, who discovered that the origin of chronic disturbances of nutrition in infancy lay not in insufficient absorption, and in secondary decomposition of the food residue, but rather in poisons, especially acids, formed

from the food materials, and from faulty function in the course of metabolism.

A further advance in our point of view had as a result the recognition of enzymes, which have proved to be common to a small extent to all kinds of milk, and to a greater extent specific for each variety, and fitted to the requirement of the particular variety of animal. The interesting researches on this subject, which belong to the last years of the nineteenth and the first years of the present century, are associated with the names of Raudnitz, Marfan, Escherich, Nobécourt, Merklen, Halhan, van de Velde, Landtsheer, Moro, Spolverini, and others. Nevertheless we are left with the impression that the significance of enzymes has been much exaggerated, and that their practical value must be but small.

Also the exceedingly significant experimental researches of Pawlow and his pupils (published in lecture form 1898) were very fruitful in connection with the proper understanding of the nature of digestion. Their results were applied by individual authors (Siegert, 1902) to the study and treatment of diseases of nutrition.

The studies of Bordet, Uhlenhuth, Ascoli, and others, on the formation of precipitin, and through it the recognition of the specificity of various kinds of albuminous bodies, stimulated the recent researches of Wassermann, Hamburger, Schlossmann, Moro, and Finkelstein. These investigators reached the conclusion that the constituents of various kinds of milk were peculiar to the particular milk. This conclusion has helped in the comprehension of certain toxic symptoms, which appear in artificially fed babies, and at the time of weaning, and frequently manifest themselves in the form of severe gastro-enteric disturbances. Indeed, an immunizing treatment is said to have been already successfully established (Schlossmann, 1905).

At the same time there have been endeavors, through researches on the microscopic anatomy of the intestinal canal, to establish findings corresponding to individual clinical types, although the respective works of Baginsky, R. Fischl, Marfan, Heubner, Bloch, Tugendreich and others have not resulted in agreement.

The sum total of the researches, carried on with the expenditure of much effort and thought, and whose most important phases I have above endeavored briefly to sketch, has not sufficed, either to clear up our knowledge of this important division of infantile pathology, or to enable us to arrange in logical sequence the several links in the disease chain. The continued failure of agreement as to the classification of the various processes belonging in this group, for which processes every author working upon the subject proposes a new scheme of classification, demonstrates clearly that our knowledge of the nature and clinical significance of the disturbances of nutrition in early

childhood has remained but a patchwork. I consider it my duty to precede the discussion of the subject with this confession.

II. GENERAL ETIOLOGY

We are concerned with the disturbances of nutrition from the food; these are manifested in a great variety of ways according to the chemical composition, biologic peculiarities, daily amount, intervals of administration, bacterial uncleanliness, and admixture of toxines. We may also include with these cases others, of infants that come into the world with inferior equipment as a result either of premature birth and the corresponding undeveloped digestive power, or of insufficient functioning power of the digestive apparatus from hereditary causes, and which even with the observance of all the precautions known to us appear to be imperiled through special susceptibility. In order to attain a physiologic basis from which we can grasp and combat the causes of these disturbances of nutrition, we must start with normal conditions, and must follow in their course of development, babies born at full term and brought up normally upon a sufficiently plentiful secretion of the mother's breast.

A digression is therefore pardonable into the subject of the physiology of the nutrition of the human infant, our knowledge of which has been built on the ground of repeated and various collected observations. The newborn infant finds in the mammary glands of its mother a fluid nutriment which is suited to its needs, and to the normal functionating of its digestive and assimilative apparatus. It is serviceable for the building up of the body substance, along the lines of normal development, that is, with a proper distribution of the growth impulse through the various organs and tissues. This fluid itself develops according to the constantly changing needs of the infant. The sucking reflex started by contact with the nipple permits it to take the food in an amount regulated by the need of sleep and the feeling of satiety, and to take in also a number of protective substances, which bestow upon it a certain power of resistance in the struggle against infections of various kinds. It takes in albuminous bodies which can perhaps be partly absorbed unchanged, but which can in any case be utilized by its body with comparatively easy chemical changes. It is able through mother's milk easily to maintain the constancy of its body temperature, to produce bowel movements in proper quantity and quality, to limit the secretion of its urine to proper amounts, to keep its intestinal flora normal, and also, perhaps, to strengthen its digestive power by means of a number of ferments peculiar to the digestion of breast-milk, and to prepare it for future changes of nutriment. Thus a fluid nourishment streams into the infant in its natural state, at body temperature, and practically free of germs. In short we see here an

example of how, everywhere in life, nature fits everything together in the smallest space and most economical way. If we are careful that the health of the nursing mother remains undisturbed, and that the taking of nourishment follows those intervals which we have discovered, from the study of the course of digestion in nursing babies, to be most favorable (literature by von Hecker, Czerny-Keller and others), then the result follows that the newborn infant by its own work causes a normal development of the breast-glands of its mother from the stage of colostrum production to that of weaning. Under such conditions we notice a steady and undisturbed development of the child, which manifests itself in a regular increase in the body weight and stature, in a corresponding strengthening of the functionating power of the various organs, and in the occurrence of walking and dentition at the proper time. Deviations from the order briefly sketched above, have as their result disturbances of the function of the digestive organs, which soon manifest their unfavorable action upon the entire organism, and which can suddenly or gradually, lead to deep-seated alterations. These find their clinical expression in the different types of diseases of nutrition.

As the fundamental principles suggest that we take normal conditions as our starting point, we shall begin with the

DISTURBANCES OCCURRING IN BREAST-FED INFANTS

In the first place the microörganisms normally present in the ducts of the mammary glands are in rare cases causes of enteric disease (Moro, von Rosthorn).

Overfeeding at the breast, through too frequent or too prolonged nursing, or both, can produce a number of disturbances, of which the symptomatology will be described later.

Febrile diseases of the nursing mother can produce injury through marked impairment of the milk secretion, changes in its composition, relapse into the colostrum stage, and excretion of infectious organisms through the milk. As to the last, the opinion of writers on this subject has recently been greatly modified, since, if infection by contact is avoided, the taking of such infected milk is regarded as doubtful (Perret).

To sum up, we must consider it assumed that the most rational procedure is that which, apart from the conditions mentioned above, entails the least danger for the function of the infantile digestive organs, and guarantees to the infant a correspondingly thriving growth. Nourishment at the breast of the mother can alone fulfil these conditions.

The next natural method of nourishment at our command is by a *wet-nurse*. This method adds a number of other possible causes of disturbance of digestion to those occurring with mother's milk. The first and most important of these comes from the fact that, if a newborn infant is put to the breast of a wet-nurse who is already in full

lactation, even with proper intervals of nursing, overfeeding with its results easily occurs. This is readily explained physiologically, since a source of nourishment which is already richly flowing is offered, instead of the normal course of development of milk production, in which the child coöperates through the sucking reflex. Thus the child, because it can get its nourishment without trouble, can easily get too much, moreover there can be no doubt that, through the close genetic relation between mother and child, the sucking power, capacity of the stomach, and resisting power of the bowel of the infant, are closely adjusted to that particular source of nourishment, which has itself grown to maturity parallel with the ovum, and which has been stimulated to its full function by the suckling's own activity. However, the disturbances caused in this way are for the most part slight, and of a transient nature, because of the great generic similarity of different human milks, it being granted that the physician in his choice takes into consideration the various existing circumstances, and finds a wet-nurse closely adapted to the requirements of the child. Nevertheless, in spite of the marked superiority of the natural method of nourishment, one should neither minimize nor entirely neglect its possible evils.

Except in the first days of life, when a very profuse flow from the breast can prove a drawback, the period of time since the nurse's confinement does not play a very important part, provided that, as is usually the case, too great differences between the duration of lactation and the age of the child are avoided. Also, to a certain extent, an adjustment takes place, since supply and demand are mutually regulated, and after a time child and nurse become fitted to each other. Indeed too much reduction of the secretion can occur, the effort for diminution going as far as drying up the breast. On the other hand, as experiences in various German Infants' Homes demonstrate, through training, a milk secretion can be attained which far surpasses the normal in amount, but which, in my opinion, always suffers in respect to the quality of the product.

The *diet of the wet-nurse* is only very rarely a cause of disturbance of digestion in the infant, and what has been said and written on this topic has been largely exaggerated. Violent assaults upon the digestive organs of a mother, or of a robustly organized wet-nurse, are required, to call forth disturbances which shall exercise their influence upon the child. I remember that during my hospital service in the Foundling Institution the diet of the wet-nurse, before the introduction of proper management, was of more than doubtful quality. Nevertheless there was scarcely ever observed any influence from this cause upon the health of the children. These observations are made, not to favor unlimited discretion, but to prevent exaggeration, and useless restrictions in the matter of the wet-nurse's diet.

It is the same way with the restriction of alcohol. In countries where the taking of weak alcoholic drinks, especially in the form of light beer, is customary, we should not uselessly stop a custom which has a favorable influence upon the appetite and temper of the nurse. The frightful pictures painted by the total abstainers with a view to teaching a fear of alcohol, are not to be dreaded. On the contrary, I would rather mention the good effects of moderate beer-drinking. Abuse of spirits, of which we have from earliest times disapproved, can cause in the child disturbance of development, and injury to health, and is strictly to be forbidden. A number of such cases have been collected by Marfan.

Also we must not allow the wet-nurse to have certain drugs, which we know from experience and from experimental research can be excreted in the milk, and which are bad for the infant. Nevertheless the number of these preparations is much smaller than was formerly supposed. We recognize as certainly excreted in breast-milk only salicylic acid, codeine, and mercury (Thiemich), while we can, as a result of animal experiments, exclude the excretion of opium, morphine, atropine, and large doses of alcohol.

Finally there remain to be mentioned those diseases of the nurse, such as active tuberculosis, severe uncompensated cardiac disease, advanced nephritis, and certain nervous disorders, which can result in such deterioration of the milk that disturbances of nutrition are to be feared. Such diseases can also cause injuries on the one hand through the danger of infection, on the other through the influence on the consciousness and intelligence of the nurse.

It should suffice to compel us to regard breast-milk as the only thoroughly suitable food, if we simply reflect that the breast-glands of the mother grow to maturity and prepare their fluid nourishment, while the fœtus is developing in utero, and under the influence of the internal secretion of the growing placenta and ovaries. This was first shown experimentally by Halban. Such teleologic conclusions find their confirmation in the observation of children who are normally born and are nourished rationally in the natural way, in the experience of farmers with suckling animals of various breeds, and in studies of the comparative nutrition of such animals. Czerny and Keller in their clear explanations were the first briefly to demonstrate the protection against disturbances of nutrition, and the very marked relative immunity attainable in sucklings through the great superiority of breast-feeding.

Everything that we know, both of the physiology of digestion in newborn infants and sucklings, and of the structure of the alimentary canal, and of the functionating power to be predicted from this structure, serves to strengthen us in this opinion.

The construction of the cavity of the mouth, with its poorly developed salivary glands, and the slight power of its digestive ferments, as well as the functional preponderance of the muscles of its floor over those of mastication proper, and the absence of teeth, demonstrates the necessity of an exclusively fluid nutriment. The full development of the sucking and swallowing reflexes at birth allow the rapid taking in and passing along of such nutriment. The anatomical arrangement of the stomach, its small capacity, its weakly developed musculature, its elastic tissue, which in the first months of life is scarcely noticeable, and is arranged only about the greater vessels (Fischl); the slight differentiation of the two varieties of gland cells, on the correctness of which I must insist, on the ground of former and recently repeated researches, in spite of observations to the contrary; the shortness of the crypts and the relatively deep extension of their epithelial layer into the necks of the glands; and finally the quantitatively slight production of a secretion of weak digestive power,—all these facts assign to this organ the rôle of a food reservoir, rather than that of a place of digestion of any considerable importance. Its function of digestion develops fully at a relatively late period, toward the end of the second year. In the suckling, intestinal digestion represents most of the assimilative function. The stomach performs but little digestive work, and therefore requires a fluid nutriment, which it can deliver over to the intestine without thorough preliminary preparation. We know, from the fundamental researches of Pawlow and his pupils, how close is the connection between the digestive power of the stomach, and of the different divisions of the intestine. We know that a normal course of gastric digestion is a necessary condition for proper intestinal digestion, and that the acidity of the chyme stimulates the great digestive glands of the abdomen to the secretion of their specific enzymes. We understand fully that any interruption in the regularity of the successive steps of this complicated process results in disturbances which manifest themselves throughout the metabolism. The relative length of the bowel in comparison with that of the whole body, the weakness of its musculature, its hardly noticeable supporting elastic tissue, its richness in lymphatics and blood vessels, its nerve fibres, for the most part without sheaths, allow it to play the part of a very sensitive organ of absorption. Such an organ can accomplish the chemical breaking up of food only when the food is presented in the form most easy of assimilation, and can utilize it to an extent which corresponds to the needs of the growing organism.

In spite of everything, *artificial feeding*,—to use a term coined by Schlossmann, although the process is really far removed from *art*,—has been more and more widely adopted. It has increased under the influence of heredity, of the passion for unlimited enjoyment of life,

of social requirements, of pernicious advice from persons around the mother, among whom midwives must be particularly mentioned, and finally of exaggeration as to how much can be accomplished by physicians with this method. We will here only refer briefly to the injuries resulting from the method of artificial feeding, which, according to general conviction, represents the most fruitful source of diseases of the digestive organs in early life.

The following figures, from both earlier and more recent times, prove that we are guilty of no exaggeration, when we assign to artificial feeding the leading rôle in the pathogenesis of such disease processes.

According to Boeckh, the former competent director of the Berlin statistical department, the mortality of artificially nourished infants is twenty times as great as that of breast-fed infants. In Munich the mortality of breast-fed babies amounted to 11 per cent., of bottle babies, 89 per cent. In Paris the figures were 18.2 per cent. and 43.7 per cent. according to Hèry. Shutt states that among 2000 cases of acute gastro-enteritis collected by L. E. Holt, only three exclusively breast-fed children were included. I will not quote any further figures, since those cited are sufficiently convincing, but will limit myself to showing that the introduction and improvement of the method of milk sterilization also, has had no noticeable influence upon infant mortality. According to Nobécourt, in all 2485 infants died of enteric diseases in Paris in the year 1885, while in the year 1899 the number was only lowered to 2106. Also Flügge could not demonstrate from statistics any diminution in the mortality of the first years of life since the more general use of sterile or approximately sterile food. Czerny's results are the same, and my own experience in the university polyclinic did not result in my perceiving any lessening of the number of gastro-enteric cases in recent years.

The next question is, What is the *nature of the damage which artificial feeding does to the digestive organs*, and what is the unfavorable action upon the general health of the infant? We will begin with cow's milk and the disturbances caused by it, as it is the most widely used substitute for human breast-milk. The causes are partly digestive, partly biologic, partly bacterial. These etiologic factors, each of which has been in turn placed in the foreground as the sole guilty one, really are interlocked with one another, and their sum total is the cause.

If we take up first the chemical differences between human and cow's milk, we find that they consist both in the quantitative percentage and qualitative structure of the several constituents. This matter has been so thoroughly treated in former books, to which I can refer, that I need touch upon it only briefly here. The higher proteid and salt content and lower fat content; the different percentage of the several varieties of proteid, the more easily assimilated proteids being present

in cow's milk in smaller amounts; the different chemical combinations of the salts, which present themselves mainly as inorganic compounds; the different composition of the fat,—all these differences explain the fact that the digestion and assimilation of cow's milk must place a greater burden upon the immature digestive organs of the human infant. In spite of all assurances to the contrary (Oppenheimer, Budin, and others), which were somewhat modified as the result of closer observation, the giving of cow's milk to an infant in the first weeks of life will always be a risky proceeding, even granted that the product is pure, and the feeding intervals exactly regulated.

The differences extend further than can be proved by chemical tests, and a number of interesting works, which comprise chiefly comparative studies of the proteids of different kinds of milk, have taught us to recognize the specific character of proteid substances. This has been established by means of Bordet's method of the demonstration of the formation of a specific precipitin in the blood (Schlossmann, Moro, et al.). Indeed, by means of a comparatively simple clinical method, we are supplied with a possible proof that the admitted difficulty of digestion of cow's milk is due to its heterogeneous character, since Moro and Gregor could demonstrate the appearance of a leucocytosis after the first administration of cow's milk, whereas breast-fed children in the height of digestion show a leukopenia. If we consider in addition the fact demonstrated by Ganghofner and Langer, that the entrance of a foreign proteid into the circulation of young children is followed by the formation of specific precipitins, and if we realize the associated processes in the organism, which, according to the prevailing theories, must precede the formation of such antibodies, we will understand completely how the giving of cow's milk even to older infants can at times be followed by severe and even actually dangerous symptoms, and how it exacts a great deal from the functioning power of the organs of digestion and assimilation (Schlossmann, Finkelstein).

A further important difference between human and animal milk lies in the fact that according to the present state of our knowledge, we are forced to administer milk in a boiled or sterilized, at any rate pasteurized, condition, a practice which in most countries should not be changed too soon. Even if we assume that the digestibility of the casein is not influenced by this proceeding, nevertheless the albumins are coagulated, the emulsion of the fat is damaged, the salts are partially freed from their organic compounds, and the power of self-protection against bacterial destruction possessed by raw proteid is lost.

Recently, attention has been directed by a number of authors to certain enzymes peculiar to fresh milk (see Marfan). Their significance, in the first enthusiasm of discovery, was greatly exaggerated, but they are nevertheless of fundamental interest, because through

them each kind of milk bears to a certain extent its vital label. Whether they play a part in digestion, such as supplementing the undeveloped digestive function of the infant, we do not know, although the results of many clinical experiences and metabolism experiments appear to be explicable by such a theory (Monrad, Hohlfeld, Cronheim-Müller). At any rate these substances are not resistant to heat, are destroyed by the usual cooking and sterilizing methods, and are weakened by pasteurization. Hence they constitute another important point of difference between natural and artificial feeding.

The increased immunity attained through breast-feeding finds its explanation in the researches of Salge, who showed that homologous albumin acts as a conveyor of antitoxin from mother to child, and also in the conclusions of Moro, who demonstrated a higher bactericidal power in the blood serum of naturally nourished infants.

The obtaining and *preserving of cow's milk* aseptically is so costly and the addition of substances such as salicylic acid, boric acid, formaldehyde, etc., to hinder bacterial development is either so ineffective, or so injurious, that we are left with nothing practical except to aim at relative sterility through boiling or sterilization. As to other methods, such as the use of ultra-violet rays (Seifert), we must wait for further experience. Does sterilization really accomplish what we expect of it? On the long road between the udder of the milk-giving animal, and the mouth of the infant, are many opportunities for bacterial contamination. Numberless organisms gain entrance to the milk, from the body of the animal itself, from its hair, from its tail, from its manure, from the air of the stable, from rubbish, from fodder, from flies, from the hands and clothes of the milkers, from straining cloths and pails, and finally from the various manipulations during transportation within and without the house. These organisms under proper temperature conditions, find an opportunity to increase immeasurably. As appears from the bacterial counts of Miquel, and of Escherich and Cnopf, they make the most thorough use of this opportunity. As a result of a closer investigation of this condition of milk contamination, physicians were brought to conceive it as the decisive cause for the high mortality statistics of artificially nourished infants, and looked to thorough sterilization for the surest means of prophylaxis. There are still at the present time enthusiastic supporters of this view.

Of the *contaminating organisms* we will for the present disregard the pathogenic varieties which excite specific diseases, and will consider the saprophytic varieties. These, on account of their different action upon milk, can be divided into two groups, the acid-forming and the proteolytic. The former cause a fermentation of the milk sugar, with the production of volatile acids, and produce a precipitation of the casein, through which the milk is coagulated. Such an

altered nutriment, even under the most poverty-stricken conditions, can hardly be utilized by the infant. Moreover the fission fungi belonging in this category are not spore forming organisms, and therefore have little resistance to heat, and are not endowed with the power of toxin formation, so that the usual heating process accomplishes the destruction of their vitality. The only question is, whether the protoplasm of these acid producers contains injurious substances which after the destruction of the germ can pass into the milk, and also whether the administration of numberless dead bacteria of this group can be a cause of danger. We still know very little about this matter, although investigations on animals by Jemma and Figari suggest that morbid symptoms referable to the digestive system may be produced in this way.

The second class of cow's milk saprophytes, which split up the proteids (proteolytic), form spores, and are consequently resistant to heat. Therefore they can grow and increase in a milk which has been freed from the acid producers by means of sterilization. We are indebted to the thorough researches made by Flügge and completed by Lübbert for our knowledge of the fact that it is not always the poisons produced by the bacteria, but also substances contained in their protoplasm, which are the active agents. Nevertheless, the clinical proof of the pathogenic action of the proteolytic bacteria in cow's milk is wholly lacking. There are certain older and inconclusive statements, which have always been cited, such as those concerning Vaughan's tyrotoxicon, which has at present a legendary celebrity. With the exception of these, we possess no clinical observations directly proving injury to the infant from the taking of insufficiently sterilized cow's milk.

Recently Escherich has called attention to the possibility of infection from cow's milk, from the observation of an epidemic of enteric disturbance, in which he suspected a peculiar streptococcus as the cause. We have also at our disposal the interesting findings of Petruschky and Kriebel, supplemented by those of Czaplewski, Rabinowitsch, and Brüning, which I can confirm from personal experience, that ordinary milk bought in the market shows in cover-glass preparations a remarkable richness in streptococci, which are not destroyed in pasteurization, and of which even the corpses can prove dangerous. Therefore we cannot exclude the possibility, that certain enteric affections can occur through cow's milk which has either been insufficiently sterilized, or in which bacterial growth has been very active before boiling. Nevertheless we lack strict proof of this.

There are also a number of reports which suggest the probability that the fodder of the milk-giving animal may have some bearing upon the occurrence of diseases of nutrition in infants brought up on such milk. This can happen in several ways. When cows are put out to pasture, the change causes looser dejecta, which afford a greater possi-

bility of infection than the more solid dejecta of the period of dry fodder. Also, certain fodder materials (brewer's grains, potato-peelings, turnip-tops, etc.) contain volatile substances which pass into the milk and irritate the gastro-enteric mucous membrane of the child. The view emphasized by Sonnenberger, that the eating of poisonous plants by grazing cows, and the passing of alkaloids into the milk is a cause particularly of very severe gastro-enteric cases with collapse, has been repeatedly disputed. It has however been corroborated recently by Piorkowski who reports that the demonstration of colchicine in the milk of such animals has been repeatedly obtained.

Also the water used in attaining a proper dilution may be of bad quality, and, through containing too much calcium oxide, nitrates, ammonia, glutinous substances, and the like, may cause injuries. Jürgensohn has recently supplied some very interesting observations on this subject.

All these facts, which I have briefly reviewed above, brand *cow's milk as a dangerous food,* especially for the young infant, and as a food which plays an important part in the etiology of the acute and chronic diseases of the infantile digestive apparatus. The most recent compilation of our knowledge of this subject is found in the book of Czerny and Keller, to which I have already repeatedly referred. This work, through original clinical observations and a thorough study of the literature, demonstrates the fact that cow's milk can be and very often is injurious, without the intervention of bacterial causes, or of biologic peculiarities, and without the passing over of poisons from the fodder, or the like. It demonstrates that certain constituents of cow's milk, in their absorption and assimilation, create disturbances in the metabolism of the child, which manifest themselves as severe injuries to its general condition, which may even threaten its life, and also act as irritative symptoms of various kinds, referable to the digestive tract. A thorough description of these deviations from normal metabolism is given in another part of this text book, and I can therefore limit myself to the consideration of as much as is necessary for the understanding of the diseases of nutrition.

The above-mentioned authors select the term *"milk-injuries"* (Milchnährschaden) for a group of digestive disturbances with well-marked clinical characteristics. They regard a too high fat content of the food as the cause of these injuries. Formerly a too high proteid content was considered the chief cause of the disturbances occurring in artificially nourished infants, and this view is still maintained by numerous authors. It is rejected by Czerny and Keller, upon plausible grounds,—an etiologic exclusiveness which is perhaps carried too far. At all events from their work, based upon the observations of a large material, it appears that cow's milk, even when properly ob-

tained, prepared and administered, can give rise to severe disturbances through the failure of a proper reaction of the infant's metabolism toward certain milk constituents.

Overfeeding, which we meet with so frequently as a cause of diseases of nutrition in breast-feeding, is easily possible in cow's milk feeding, as in all forms of artificial rearing. It is possible, since, as pointedly expressed by Epstein, "gate and market stand open." In spite of all our care as to quantity and caloric value, the efficiency of our precautions is so impaired by the variations in the amounts of nutritive substances found in our market milk, that overfeeding is hard to avoid. The bad effect of overfeeding is increased by the fact, that with the use of less easily digested food an atonic condition of the stomach soon develops, which leads to a delayed emptying and consequent stagnation of the gastric contents (Pfaundler).

The milk of other animals, such as ass's, mare's, and goat's milk, is not of much practical importance in this country, since these animals are used but little in the farming and dairy industries, and their milk is rather costly. The reports from German clinics and French hospitals of goat's and ass's milk give results which are very variable but on the whole rather unfavorable, and this method of feeding, even with direct application of the child to the nipple of the animal, offers no protection against diseases of nutrition (Marfan, Klemm, Schlossmann, Ranke, Czerny, Brüning).

In many countries the feeding with starchy foods in the early weeks of life is much practiced. These foods, whether in the form of cereal jellies, gruels, or various commercial infant foods, constitute a further cause of severe and even fatal diseases of nutrition, with acute or chronic course. Czerny-Keller, to whom we are indebted for the exact study and proper appreciation of the value of these disturbances, designate them by the striking name of "*starch-injuries.*" I shall speak later about the symptomatology of these conditions, of which I still have a vivid remembrance from my service as assistant at Munich. These very disturbances demonstrate that methods of nutrition based upon theories or upon the results of metabolism experiments can suffer bad shipwreck in practical application. Although we have been furnished with metabolism experiments (Carstens, Heubner, et al.), which seem to demonstrate the value of certain starches in early life (to which however the different results of Schlossmann are opposed), nevertheless the method often fails us in practice.

Similar dangers are incurred much less frequently by the early use of other foods, particularly eggs and meat, which usually produce severe disturbances mainly localized in the large intestines. Particularly the idiosyncrasy toward eggs in any form manifests itself in many children with the clearness of a scientific experiment. After the admin-

istration of the smallest quantity there can appear fever, foul diarrhœa, urticarial eruptions, and nervous disturbances. These symptoms, since they show a great similarity to the results of the injection of animal blood serum, are suggestive of poisoning by foreign albumin (Bendix, Finkelstein, et al.).

As to glutinous substances, Gregor has demonstrated their action in producing diarrhœa by special nutrition experiments, while Czerny-Keller report similar observations after feeding with a nutriment rich in glutens, as, for example, soup made of veal bones. They designate these disturbances "*gluten-injuries.*"

There is a critical period in an infant's life, during which the influence of the nutritive injuries sketched above is of special meaning and importance. Such a period is represented by the first weeks of life, during which most children react with severe symptoms against every form of artificial nourishment. If this stage is survived, or if the disturbances do not run an acute course, then deep-seated injuries of a chronic nature are often produced, which in their results dominate the development of the child during the entire period of infancy and often long afterward.

I shall consider next the so-called *mixed feeding* ("allaitement mixte"), which consists in the simultaneous administration of both natural and artificial food. This proceeding is in wide-spread use, for example, here in Bohemia, particularly among the Slavic population. With mixed feeding, diseases of nutrition appear less frequently and in milder form, than in the weaning period which constitutes a second critical time in the life of the infant. Observations of this kind, on the one hand are suggestive of the action of the enzymes of human milk, particularly its peculiar amylase, in strengthening digestion, and on the other hand they are suggestive of the injuring of the bowel by a foreign albumin.

In the most diverse countries and parts of the world there occurs a very marked increase in the *infant mortality in summer.* This increase, which is due to the frequent occurrence and severe course of diseases of nutrition, deserves a brief discussion here.

There is no doublt of this fact, as it appears in all official mortality statistics with striking clearness. Even in localities where this is not so convincingly the case, as for example in Prague, where I have been able to demonstrate the absence of a maximum mortality in summer, a close study of the figures explains the reason of the exception. In these cases there is an artificial alteration of the conditions because the infants born in institutions, whose increase dominates the birth statistics, depart for the most part after a few days into the provinces, with a consequent reduction of the infant mortality. It is further established that in states and countries the population of which mainly

nurse their babies, the mortality of infancy does not attain a clearly marked maximum in summer. On the other hand, in countries where there is much artificial feeding, the summer maximum is very high. If the population is considered according to wealth, taking the size of the dwelling as a standard, then it appears that the highest summer mortality of infants prevails among the poorer classes, a fact which is true among both the races which nurse, and those which do not nurse their infants. In hot weather, there occur digestive disturbances of a specially severe type, while in the course of chronic affections of the digestive tract, acute exacerbations occur, which may lead to a fatal end. There must be circumstances of special significance to explain these facts.

Under the influence of a high temperature outdoors, milk spoils easily, especially in the wretched quarters of the poor. This has been advanced as the chief cause of the summer cases, an opinion which has much in its favor. It does not explain everything, for it does not affect, for example, the summer gastro-enteric cases occurring in children fed on breast-milk only, or on properly sterilized cow's milk. Others assume as a cause that the heat so alters the digestive function, that an increase occurs in the virulence of the bacteria which have hitherto existed normally in the intestine. This is difficult to prove, and is not yet proven.

The relation between infant mortality and the state of the surface water is entirely beside the point, as it has nothing to do with the etiology of the summer diarrhœas.

Th. Meinert has expressed the opinion that the cause lies in overheating, a process analogous to the heat strokes of adults, an opinion which is supported in one of the recent publications by Illoway, an American author.

It is entirely certain that we cannot explain the causes of the regular appearance of summer diarrhœa, although it is an undoubted fact that the character of the food and sanitary surroundings have an influence upon the summer mortality of infants from diseases of nutrition. This fact suggests that cleanliness and digestibility of the food, as well as careful hygiene, play the chief part in prophylaxis.

We should next consider the possibility of the acquirement of diseases of the digestive system by means of contact infection. This results in an endemic prevalence of enteric troubles particularly in places where infants are crowded together, as in nurseries, foundling institutions, infant hospitals, and the like. Such epidemics have been reported in recent years from various localities, and have thrown new light on the prophylaxis and care of infants in institutions (Escherich, Heubner, Finkelstein-Ballin, et al.).

We have passed in brief review the various causes which can produce diseases of nutrition in infancy. It remains, in concluding

this part of the subject, to speak of the *bacterial causes which play a primary or secondary part in the etiology of these diseases.*

We are indebted to the work of Escherich, Tissier, and Moro for an apparently accurate description of the normal flora of the infant's intestine. The value of these results is somewhat limited by the fact that they are conclusive only with respect to the organisms present in the lower part of the intestine. The thorough study of the vital conditions and probable significance of these organisms in the course of normal digestion affords us a hope, that we shall soon be able to understand their pathologic variations and their significance in pathogenesis. This has always been our pious desire. As suggested by Schmidt and demonstrated by Strassburger and others, comparative investigation of the intestinal content in stained cover-glass preparations, and on culture media, prove that a great many bacteria which can be demonstrated by staining can not be grown in cultures. Consequently the very foundations of research are not entirely solid. Very few processes have succeeded in finding bacteria which, by reason of almost exclusive recognition in the stools, penetration into the body, microscopically demonstrable connection with the intestinal lesions, pathogenicity toward animals, and the serum reaction, can be considered as actually proven causative. This holds good for hardly more than streptococcus enteritis (Escherich, Hirsh, Libmann, Spiegelberg, et al.), colicolitis (Escherich), and pyocyaneus infections (Nobécourt). To explain the findings in other cases it is necessary to assume the symbiosis of several varieties of organisms. In still other cases, in which the normal bacteria were present in cultures in more or less purity (Baginsky, Booker), nothing better remains than to have recourse to increase of virulence (Lesage), formation of soluble poisons (Zaborsky), formation of food-decomposition products irritative to the intestinal mucosa (Baginsky), and other forced and badly proven explanations.

Etiologic investigation has made further progress through the results of collective bacteriologic work in America, which have been reported by Flexner and Holt, and confirmed by the researches of other authors (Leiner, Jehle). From these results it appears that in the majority of cases of summer diarrhœa it is possible to obtain from certain parts of freshly passed stools by certain special methods of cultivation, various types of dysentery bacilli, which are also found to some extent in the normal stools of healthy infants. There is considerable ground for believing that further studies in this direction will lead to a substantial change in our pathogenetic point of view.

This should also result from a refinement of our culture methods, particularly perfecting of the technique of growing anaërobes, which will probably give unexpected results (Passini).

The bacterium coli, which formerly occupied the foreground, and was considered the exciting cause of all possible pathologic processes, (a view which, as I pointed out a number of years ago, was not sufficiently proved) has now retreated somewhat into the background, because the total armament or modern bacterial diagnosis, such as serum reaction, formation of flagellæ, and so forth, has not been conducive to increasing its reputation (Escherich, Pfaundler, Nobécourt, et al.).

A great and lasting service was undoubtedly performed by Czerny, Keller, and the other active co-workers of the Breslau children's clinic, when they forsook the one sided bacteriologic standpoint in their studies, and elucidated the important bearing of disturbances in the processes of metabolism upon the pathology of diseases of nutrition. I shall refer to the detailed researches of these authors in another part of this work. If they also proceeded at first in a somewhat one-sided direction, and gave to an ambiguous and inconstant finding too marked a significance, they nevertheless furnished us with valuable insight into the mechanism of assimilative processes, and established a better understanding of the origin, and a rational standpoint for the therapeutics of the diseases of nutrition. We shall speak more thoroughly of all this in the appropriate chapter.

III. CLASSIFICATION OF THE DISEASES OF NUTRITION

The foregoing discussion on etiology and pathogenesis has demonstrated that our points of view are in a transition stage, and have attained no definite clearness. A survey of the attempts at classification of the diseases of nutrition by various authors offers a still more unpleasant perspective.

The older attempts at classification, although they lacked a pathologic, bacterial, and chemical foundation, and were based entirely upon the rather vague symptomatology of these diseases, nevertheless succeeded, through the masterly power of observation of the investigators, in defining particular types, which we must recognize even to-day on account of their definite clinical characteristics, and which must form the basis of any classification based upon our broader knowledge of pathogenesis. Passing over these older labors, we must next consider von Widerhofer's classification, which is based upon the anatomical findings. Although this author in his discussion of diseases of the stomach (of which he describes no less than 15 different types), sticks pretty closely to post-mortem findings, yet in gastro-enteric diseases he has to fall back partly upon the clinical course, and speaks, among other things, of dyspepsia, cholera infantum, and so forth. Anatomic research has thrown valuable light on the subject of infantile pathologic anatomy, which I shall discuss thoroughly in the next chapter, but it can not be used as a basis for classification of this group of dis-

eases, because the anatomic types corresponding to the particular forms of disease are not sharply enough outlined. Another point against such a classification is the fact that frequently the severest clinical symptoms give negative, or almost negative, post-mortem findings.

For this reason Baginsky, who among modern writers places the highest value upon the anatomical findings in gastro-enteric affections, has been compelled to forsake this basis in his attempted classification. He, like so many others, has not remained true to his original scheme, so that with respect to this matter inconsistency is the rule, Thus to Escherich we owe three distinct systems of classification, differing not only in the number of forms described, but also in essential principles; Lesage advances a new proposal in almost every publication; and we find in the various French monographs wholly diverging classifications which are based at times upon bacterial etiology, and at other times upon clinical standpoints (Marfan, Rothschild, Nobécourt, Ardoin, etc.). Actual progress was made when Heubner first sharply differentiated the various forms according to the methods of feeding the infant, which differentiation is accepted by Czerny and Keller in their text book.

Since our knowledge of the nature of the various diseases of this class is still in process of development, it is for the present impossible to take etiologic standpoints as the basis of a principle of classification, even when mixed with clinical facts. The idea of "digestive infection," as conceived, for example, by Lesage, Thiercelin, and others, has a significance only for that class of cases in which we can demonstrate with certainty the action of organic exciting agents. But there are a number of processes, in which parasites of endogenous or exogenous origin play either no part, or a very unimportant one. Nevertheless such processes must also be given a place in a scheme of classification. We must require of our scheme that it will enable us to diagnose correctly through our clinical methods every disease met with clinically, except rare and exceptional cases, and to place it under its proper heading. Further difficulties are encountered in the multiple interrelation of the processes, by which a gastro-enteric affection of acute onset may end in a chronic stage, or vice versa prolonged disturbances of digestion may undergo acute exacerbation. Also, as often occurs in such combinations, the imperceptible transition of individual types into one another, and the successive involvement of the various divisions of the alimentary canal, are further sources of confusion. From all these circumstances the boundary lines must remain movable, and yet find their expression in the classification scheme. Taking these considerations into account, Czerny and Keller have divided the diseases into three great groups, which they designate: (1) disturbances from alimentation; (2) disturbances from infection; (3) disturbances from congenital defect in the constitution and body structure. They add

the proviso that a child can at one time manifest disturbances which belong in several of these groups. But their further attempt to subdivide the disturbances from alimentation into milk-injuries, starch-injuries, proteid-injuries, and gluten-injuries, seems to be too schematic, because the individual components of the food do not manifest their injurious action with a clearness sufficient for the construction of a definite clinical picture. Also the efficacy of a special feeding therapy, based on diagnosis, in allaying the corresponding disturbances must not be given too one sided a significance, since other factors such as restriction in the amount of food, proper feeding intervals, and general hygiene of the child are operative in producing the result. Moreover, too sharp a division of cow's milk injuries according to their bacterial and chemical nature cannot easily be applied in practice. On account of the great individual differences in the reaction of different children, we can never be sure what bacterial content in milk can be borne by the child without result, and consequently cannot tell whether the disturbances present are to be attributed to bacteria, or to insufficient absorption of the fat, carbohydrate, and so forth.

If I also undertake to propose a grouping which shall enable us to arrange the affections met with clinically in definite categories I do so in the full knowledge of the inadequacy of any such attempt, because we still have no conclusive knowledge of the nature of the disturbances of digestion and nutrition in infancy. I propose the following classification:

I. Disturbances of Nutrition in Breast-fed Infants.

(*a*) From overfeeding.
(*b*) From insufficient food.
(*c*) From unsuitability of a special breast-milk.
(*d*) From insufficiency of the digestive organs (may result from premature birth, hereditary taint, intra-uterine infection, malformations of the digestive apparatus).
(*e*) From bacterial contamination of the food.

II. Disturbances of Nutrition in Artificial Feeding.

(*a*) From overfeeding.
(*b*) From insufficient food (too great dilution).
(*c*) From failure of utilization of the food (either as a whole, or in its individual constituents).
(*d*) From bacterial contamination.

It is self-evident that in every child the different causes specified in the above classification can be combined. We can designate disturbances according to their course as acute, subacute, or chronic, the last being at times interrupted by acute exacerbations. A differentiation

according to the exclusive or prepondering involvement of the particular parts of the bowel, does not seem to be serviceable, because, as a result of the intimate functional connection, the different parts of the digestive tract are involved at the same time, or in quick succession.

Indeed in this classification we must abandon old established terms, such as dyspepsia, cholera infantum, follicular enteritis, and must also strike out atrophy as an independent disease conception. We can do this without hesitation, because in the above classification, the characteristic outlines of the disease picture on the one hand find their place in the symptomatology, and the analogous results of different causes, on the other hand, are not erroneously brought together in a general clinical type.

IV. PATHOLOGIC ANATOMY

I have already mentioned in the preceding chapter, that we can not establish specific post-mortem findings, which correspond to and are produced by definite forms of diseases of nutrition. These diseases often show a lack of correspondence between the clinical symptoms and the results of pathologic investigation. For these reasons a condensed discussion of the pathologic anatomy of the diseases seems advisable.

Those who have an opportunity of performing many autopsies are frequently astonished to observe that the severest clinical gastro-enteric symptoms with widespread manifestations may show at the autopsy table such slight lesions that the pathologist is in actual perplexity as to what he shall assign as the cause of death. This is true especially of diseases of nutrition with very acute course, in which the post-mortem changes may be limited to passive congestion in certain regions, as meninges and brain, hypostatic congestion in the lungs, and slight swelling and punctate reddening of the mucous membrane of the stomach and intestine. In rarer cases this redness is more marked. The contents of the various divisions of the intestine are variable in their appearance, and in places scarcely deviate from the normal. They do not often show the reddish coloring and flocculent admixture characteristic of true rice-water stools.

Older observers, as H. Schwartze, Müller, and others, have called attention to this peculiarity of the post-mortem findings, which they attribute to the action of soluble poisons. Where true choleriform symptoms have been present during life, the general appearance of the cadaver usually reveals the fact. The depressed fontanelle, the deeply sunken eyes, the overlapping of the cranial bones, the pointed nose and chin, the loose wrinkled skin of the extremities, especially of the thighs, the indrawn abdomen, showing on its surface greenish discoloration only a few hours after death, and the half-flexed position of the arms and legs, all appear as signs of the severe course of the disease.

Upon *opening the skull,* one often finds in such cases, in addition to the hyperæmia mentioned above, sinus thrombosis of varying extent, œdema of the brain substance, a slight and usually reddish colored exudate in the ventricles, less often seropurulent, or purulent inflammation of the pia mater, or of the cerebral tissue itself. Upon opening the middle ear can be found fairly constantly an accumulation of pus. When death has occurred very rapidly, with profuse vomiting and diarrhœa, and alied symptoms (cooling of the body and a peculiar hardening of the skin), the brain appears notably dry, its substance is thickened, and the pia mater is of a peculiar adherent character.

The *mucous membrane* of the mouth and pharynx is swollen, reddened, and dry. In very young children a profuse growth of thrush is often found, which covers the dry, leathery, and brownish tongue, the gums, the inner surface of the cheeks and lips, and the soft palate, and extend downwards, occasionally deep into the œsophagus, rarely even into the stomach (Parrot).

The *lungs* are very hyperæmic in their dependent portions, and on section exude a reddish foamy secretion. More rarely, thickened areas may be found, varying in size from a pea to that of a hazel-nut. The pleuræ show little scattered ecchymoses, and, in consequence of the marked loss of water before death, are dry and leathery, and occasionally also the seat of purulent inflammation.

The *cavities of the heart* are filled with dark blood clots. The heart muscle shows no notable macroscopic changes.

On opening the *abdomen,* the appearances vary according to the length of time since death. If the autopsy is made shortly after death, the bowel is usually found collapsed, and its contents are either watery, occasionally resembling rice-water, or else greenish yellow mixed with white lumps. The stomach as a rule is empty and contracted. If a longer time has elapsed after death, the intestinal loops appear distended with gas, their outer surface is occasionally reddened, and the stomach also shows some distention. Its mucous membrane, and that of the whole bowel may appear completely pale as if washed out, and show no trace of catarrhal swelling; more frequently, however, it appears slightly swollen, somewhat œdematous, injected in spots and streaks, showing at times little hæmorrhages and erosions, or, with more profuse hæmorrhage, dark brownish streaks. Corresponding to this condition there is either no intestinal contents, or a little mucus, or dark brown masses resembling coffee grounds floating in cloudy fluid.

Except in the cases already mentioned in which there are no macroscopic changes, the mucosa of the small intestine is traversed by tree-like branching vessels filled with blood,* or else relaxed and

* See Plates 41 and 42 for the picture of such a condition taken from a Kaiserling preparation.

œdematous; at times, especially on top of the fold it is hyperæmic; more rarely hæmorrhagic or eroded.

The *peritoneum* participates in these changes at most with a slight injection, which can spread entirely irregularly over the bowel, but which usually shows its greatest intensity in the lower part of the ileum, and in the ascending and transverse colon, and is sometimes widespread and sometimes confined to little areas. The follicles appear to be involved to a varying extent. Recently Ruf and Tugendreich have correctly pointed out, that the finding varies according to the stage of intestinal digestion at the time of death, and that we are not familiar enough with the normal condition of the follicles, such as would be found for example in healthy children suddenly dying from accident, to be able properly to call their swelling pathologic. The fact is, that at times the mucous membrane of the large intestine is found looking as if strewn with white sand, in other cases the prominent solitary follicles appear surrounded with a circle of vessels, or else involvement of the follicles in the changes of the mucous membrane may be entirely lacking. No regular rule, no relation to the clinical symptoms can be established. Often erosions or ulcerations of the solitary follicles are found, while Peyer's patches are hardly involved in the process, or at least show only slight swelling, which may perhaps be considered digestive.

Also the *mesenteric lymph-nodes* appear normal, or slightly enlarged and a little reddened on cross-section.

Changes are almost constantly found in the *liver*, varying from slight swelling and passive congestion to notable enlargement, with punctate or diffuse pallor, or with yellowish coloring. There may be in the latter case either increase in consistency, or friability with a fatty cut surface. Also here the findings show such an absence of regular rule, that they bear no relation to the clinical course (Terrien). They only permit one to say that there must have been a certain duration of illness, in order to produce such marked parenchymatous or fatty degeneration.

The condition of the *spleen* is also rather variable. Cases with rapid course and severe clinical symptoms show an acute infectious splenic tumor, cases in which the tissues have lost much water show a small spleen with wrinkling of the capsule, while the most common finding is an organ of normal size and consistency with marked passive congestion.

The *kidneys* are practically always involved. They are enlarged and pale with markedly injected glomeruli; the cortex appears streaked with red or entirely pale, and swollen on section; the pyramids are very hyperæmic; and the pelvis and calyces are usually injected and secrete a cloudy fluid. A not infrequent finding in severe cases is thrombosis of the renal veins with its resulting secondary appearances.

So much for the macroscopic findings in cases running an acute course with rapidly fatal ending. If the process becomes prolonged,

or passes over into a chronic stage, then the reaction of the mucous membrane and organs is the more intense, and the more appreciable on post-mortem examination. Mentioning only essentials, we find on the one hand that the stomach is dilated, with its walls thinned, its greater curvature reaching down to the umbilicus or still lower, its mucous membrane much thinned as if macerated, and its cavity filled with grayish white fluid in which float large or small curds. On the other hand the organ may appear contracted, its mucous membrane feeling infiltrated and thickened, especially near the pylorus. The mucosa is also much wrinkled, and, on top of the folds, injected, with hæmorrhagic or brownish discoloration, at times even necrotic. The intestine, on opening the abdominal cavity, presents a degree of distention which varies with the period of time between death and the beginning of the autopsy. The large intestine, particularly its transverse portion and sigmoid flexure, fills most of the anterior abdomen, and appears slightly dilated, thin-walled, and much bent and twisted. The jejunum and ilium appear almost covered by the colon, and, assuming an early autopsy, show little distention, and in many parts even complete contraction. During my service as assistant in Munich, when I had frequent opportunity both in the Children's Hospital itself, and in the different morgues of the city to perform autopsies on children dying of chronic diseases of nutrition, especially such as result from too early administration of starchy food, I was frequently struck by the length of the vermiform appendix in these children and by the marked distention and lengthening of the large intestine. I induced Klaus, who was at that time in charge of the children's clinic, to undertake comparative measurements of the length and circumference of the bowel in naturally and artificially nourished children, because I knew that the frequency of intussusception and volvulus among the Russian population is to be traced to an increased length of the bowel and mesentery caused by the preponderating vegetable diet of the poorer classes, and that therefore the influence of diet upon these conditions was already established. As Klaus left Munich shortly afterward, and as I returned a few weeks later to Prague, where I found no opportunity for making measurements, the question remained unsolved. It was all the more interesting to me, when a publication by Marfan appeared a few years later, in which these changes were emphasized, and in which in cases of this kind a very notable lengthening of the bowel was reported, as much as twelve times the body length, or double the normal. This lengthening affects both the small and large intestine, although it is more marked in the latter, and is usually accompanied by dilatation. It is well to establish these facts here, although they will be met with again in speaking of Hirschsprung's disease (megacolon congenitum).

The mucous membrane of the duodenum and small intestine ap-

pears thickened in places, with injected portions alternating rather regularly with pale areas. The follicular apparatus appears involved to a varying extent, from slight swelling to marked infiltration of the solitary and grouped follicles. These at times show only projecting grayish yellow patches, or lenticular nodules, and at other times they show a grayish slate coloring or marked hyperæmia, and are surrounded by a circle of vessels. These may present more or less deep ulcerations, which finally become confluent, and lead to the formation of an irregular ulcer, with a purulent or membranous base. I have had reproduced some microscopic preparations and photographs from the Gratz Children's Clinic showing these types.* In the Peyer's patches this inflammatory process is limited to infiltration, or occasionally hæmorrhage, while necrosis with the resulting ulceration and confluence of these ulcers is found only in the solitary follicles of the large intestine. Where most marked the process passes over without any sharp dividing line into the anatomical picture of infantile dysentery, which is caused by bacteria, and the boundary lines of which are also shifting, as the most recent reports of Jehle demonstrate.

There are cases in which the pathologic changes in the large intestine are to a certain extent characteristic, in that they occur with little or no involvement of the other divisions of the intestine. Consequently the establishing of the anatomical conception of colitis (enteritis follicularis) is to a certain extent justified.

The serosa is usually not involved, except that with intense inflammation of the solitary follicles with ulceration, the process can extend to the peritoneal covering. Also the mesentery shows at most only slight swelling and induration of its lymph-nodes, while its peritoneum remains unchanged.

The other organs are affected in a varying degree. The liver presents almost constantly a general enlargement. One finds at times a more marked extension of the process of fatty degeneration and infiltration already described in the cases of acute course, or else the tissue shows only here and there lighter spots, being otherwise of normal or darker color and harder consistency.

The same is true of the kidneys, which consequently give a clear impression of induration, and of the spleen, which usually shows chronic enlargement and induration.

When the symptom-complex is clinically that of *atrophy*, which can form the starting point of various types of acute and chronic diseases of nutrition, the cadaver presents the picture of marked emaciation. The skull appears small, its bones overlapping, the face senile and shrunken, the lower jaw sunken, the neck thin and wrinkled. The

*See Fig. *l* on Plate 42 and Figs. I, II, III, Plate 39.

skin of the extremities is loosely hanging, shrivelled, and entirely without fat, and is often the seat of various suppurative and ulcerative processes, while the skin of the abdomen in contrast to the general emaciation is tight like a drum. Small nodules shine through the greatly thinned skin of the anterior abdomen, which appear to be connected with thin bands of fibrous tissue. Opening the skull causes a very slight effusion of thin pale blood. The meninges and the veins of the convexity appear thinner than normal, and almost empty of blood, and the brain substance itself appears dry and extremely pale. Infrequently, and as a rule only in infants which have died with the symptom-complex of atrophy in the first three months of life, are found suppurating processes in the meninges, cerebral substance, and the accessory cavities of the skull. This process when present in the brain substance, consists of multiple pus cavities irregularly scattered through the tissue, or else of diffuse hæmorrhagic and purulent softening of the tissue.

The lungs are usually the seat of inflammation which presents itself at times as chronic purulent bronchitis, at times as lobular pneumonic areas, often becoming purulent or gangrenous, at times as a lobar infiltration, and which in the pleura presents itself as serofibrinous or purulent inflammation.

The intestines and stomach show notable distention, and a thinning of the walls to such an extent that their contents are often visible. On the surface are seen only a few scarcely filled vessels. The mucous membrane appears in places as thin as paper, in other places of normal thickness, but is always markedly pale and looks washed out.

The large abdominal glands show shrinking and increase in consistency as a part of the general wasting, and only exceptionally show suppurating areas. On the other hand such areas are very commonly present in the subdermal cellular connective tissue, and appear in the form of abscesses which are localized sometimes in the superficial and sometimes in the deeper layers, and which contain thin greenish yellow pus. Also one finds not infrequently, especially in very young infants, ulcerations of varying extent, usually resulting from inflamed parts of the skin. They may occur in many places, as the back of the pelvis and heel, and may go as far as to lay bare the bone.

The *histologic changes* in the different types of acute and chronic diseases of nutrition may be treated as a whole, since they usually represent only different grades of intensity of the same process, which localizes itself with special clearness at times in one kind of tissue, at other times in another. A change of opinion has taken place in the course of time which has replaced the original undervaluation of the importance of the histologic changes. Improved technique, and the observing of certain precautions has taught us certain sources of error, and made us more careful in judging and interpreting many

findings. Among these precautions I must mention as of first importance, the examination of material either absolutely fresh, or obtained soon after death, and the taking into consideration of the stage of digestion at the time of death, and of the amount of contraction of the bowel. If we allow for all this, and proceed with the greatest precautions against drawing false conclusions, we must still admit that the microscopic changes form an integral element of the pathologic process, and help in the explanation of the individual phases of its clinical course. This I hope to be able to prove, by means of numerous histologic pictures, for the most part especially prepared for the purpose of this article.

The lesions are seated in the superficial epithelium, the gland cells, the interstitial tissue, the follicular apparatus, and the vessels, in every possible combination of involvement. The superficial epithelium covers the inner surface of the stomach and entire intestinal tract without a break, as I have demonstrated and Reyher has confirmed, in contradiction to the observation of Disse. On this superficial epithelium occur necroses as a result of capillary hæmorrhages (Bloch) which create small areas of loss of substance. These lesions can attain important significance on account of their multiple and widespread occurrence as well as through the destruction of the normal defence against the bacteria always numerous in the mucous coating covering the mucosa (Marfan and Bernard) and also through the alternating of absorption conditions (Fig. *m* on Plate 42 and Fig. II, Plate 40). The coating over of the epithelium with mucus is regarded by Heubner as the expression of a process of defence against toxic irritation of the inner bowel surface, while Tugendreich disputes its pathogenic importance, and, on a basis of researches on the intestines of young dogs, regards it as a normal appearance connected with the physiologic process of digestion. It is a universally established fact, that the severest diseases of nutrition of long duration leading to atrophy do not necessarily affect the superficial epithelium at all, so that in fresh specimens it presents a normal appearance in all the parts examined. On the other hand there are processes of acute and chronic course, which leave behind their traces in this tissue-layer, and lead to destruction or a peculiar swelling of the epitheluim, obliteration of the cell walls, and destruction or difficult staining of the nuclei. In these cases the microscopic picture suggests the action of a severe poison, and finds an analogy in the lesions of experimental poisoning. On the basis of my own researches with material carefully obtained, preserved, and treated, I must mention a destruction of the superficial epithelium occurring over wide areas, with subepithelial hæmorrhage, necrosis of the deeper tissue, and fibrin formation in and around the area of loss of substance. Examples of this are to be found among the microscopic pictures (Fig. *h* on Plate 41).

I have observed a peculiar form of swelling in the gland cells of the stomach, which I consider a coagulation necrosis. This is also shown in the illustrations. Marfan and Bernard describe similar changes in the gland cells of the stomach and intestine as a mucoid degeneration, and demonstrate their mucous character by appropriate staining methods. There is also a preparation exemplifying this (Fig. I, Plate 40).

Only further investigation with different methods of preservation will show to what extent vacuole-formation in the intestinal epithelium represents an artificial finding, because in alcohol preparations the fat drops are dissolved and leave in their place vacant spaces.

To sum up, there can be no doubt according to the present stage of our knowledge, that, especially in the course of acute diseases of nutrition, there can be observed a number of significant lesions in the superficial and glandular epithelium which have certainly developed during life, and which can not be without importance for the function of the mucous membrane.

Baginsky has described hypertrophic processes in the crypts of Lieberkühn, through which the crypts on the one hand grow up above the level of the mucous membrane, and on the other hand break through Brücke's muscle (muscularis mucosa) and sink into the submucosa. Cysts result from this, through coalescence of several dilated tubules. Heubner considers such a picture due to oblique sections. Tugendreich, who recognizes the cyst-formation, finds the downward growth of the glands into the submucosa only in places where there are follicles, as there the Brücke's muscle is deficient, and the glands can easily penetrate into the soft follicular tissue. I freely admit this latter possibility, but my own preparations, one of which is reproduced here (Fig. *d* on Plate 41) lead me to consider Baginsky's observation entirely correct. Also cysts can be found without this hypertrophic process, as is shown in one of the preparations reproduced here, in which cysts appear in the midst of the regular parenchyma of the crypt, and are to be explained by retention of secretion. In some places are seen swollen gland cells which appear partly necrotic, near which are clumps of microscopic networks of fibrin, and masses of mucus. In other places the swollen cells have entirely disappeared, leaving only homogeneous lumps, while a flattened cylindrical epithelium lines the cyst. In the neighboring cysts are seen similar lesions in the cells, a deep extension of the mucous plug down to the bottom of the gland, and bands of mucus, which last also invade the free surface of the mucous membrane.

The vessels present in the more acute cases the picture of very marked hyperæmia, both in the mucosa and submucosa. One frequently sees diapedesis of red corpuscles into the tissue, formation of larger hæmorrhagic areas, and consequent destruction of specific tissue

elements. The more chronic the course of the disease, the less marked becomes the hyperæmia, to be replaced by thickening of the arterial walls, and accumulation of inflammatory cells about the vessels.

These latter lesions seem to form the starting point of an interstitial overgrowth, which is not notable in the acute cases, but which in chronic cases forms the leading feature of the picture. It forces apart the crypts, pushes its way through the tissue of the villi, strangulates the neck of the glands, thus constituting a further cause of cyst-formation, notably increases the thickness of the mucosa, and often densely infiltrates the submucous tissue. I believe that we have every reason for assuming that such a very marked and widespread process in the intestine must have a notable influence upon the process of absorption by the mucous membrane. In cross-sections can be seen the multiform ways in which the entangled crypts are distorted and compressed (Figs. *i* and *a*, Plates 41 and 42).

As to the distribution of the lesions described above, Bloch, who has made a topographic investigation of numerous divisions of the intestine, has stated that apart from the processes localized in the stomach, the lesions reach their maximum in the region of the cecum, and become less and less above and below the point. This agrees with the fact that in this region the intestinal contents remain longest, and thus can exercise most intensely their injurious action.

The presence of Gram-staining and Gram-decolorizing micro-organisms in the mucous layer covering the mucosa and in exudate less rich in cells, is an almost regular finding. They are mainly in the supra-epithelial layers, and not to be found either in the lumen of the crypts or in the deeper layers, or inside the lymphatics and blood vessels (Fig. *k*, Plate 41). But in certain cases, with carefully obtained and preserved material, they can be found in the crypts, in the interstitial tissue, in the region of the follicles, in the blood vessels and lymphatics, and even as far as the serosa. Typical lesions of the tissue show that these organisms have entered these regions during life, and have left behind their traces. The possibility of a general infection of the body originating there must be admitted, although I can give the assurance from my own wide experience that such an occurrence is very rare.

I have repeatedly mentioned that in the severest disturbances of nutrition, those of chronic course resulting in atrophy, the intestinal canal shows very slight anatomic changes, and that, because of this almost negative histologic finding, a disturbance of the processes of assimilation has been assigned as the cause of this severe symptom-complex. Baginsky alone expresses the opinion, based on his microscopic researches, that atrophy is due to a destruction of the absorbing tissue elements over a wide stretch of intestine, and a resulting progressive cachexia from insufficient taking up of the food by the tissue.

Heubner considers that Baginsky's findings are erroneous, and that they are to be explained through the investigation of much dilated parts of the bowel, in which the marked stretching apart of the villi and crypts gives a false impression of their disappearance. Gerlach, Habel, Kuskow, Bloch, and Tugendreich, agree with Heubner. They made comparative preparations of adjacent contracted and dilated parts of the intestine, and demonstrated that in the former the structure of the intestinal wall was wholly intact. Bloch brought a new fact to light. He called attention to peculiar cells, situated in the fundus of some of the tubular glands, particularly in the region of the large intestine. These were first described by Paneth, and can be distinguished by proper staining methods from the adjacent cells, on account of their different nuclear content. Bloch noticed in cases of atrophy a marked diminution in their number, and through this he explains the deficient absorption in these conditions. This matter is still unsolved, and we must await a further confirmation of Bloch's conclusions. Tugendreich failed to find any such confirmation in the cases he studied. I for my part willingly admit the explanation for this condition in a stretched intestine, as given by Heubner and the other writers named above, of the correctness of which one can easily convince himself (see Fig. V, *a* and *b*, Plate 40). I should like to raise the further question whether this very marked and extensive stretching of the bowel in atrophic children with pushing apart of the villi and glands, may not also in itself be of significance in absorption. We must take into consideration the clinical experience which teaches us that the absorptive power of such patients frequently improves contemporaneously with diminution in the prominence of the abdomen, and that we see in this diminution a favorable prognostic sign. It would be too great a depreciation of the value of the anatomical finding, if we should have no faith in the significance of these effects of stretching.

We will next take up briefly the histologic lesions in those organs which deserve our attention on account of their anatomic position and functional relation to the alimentary canal. Little has been reported on the macroscopic appearance of the *mesenteric lymph-nodes*. My own observation in cases in which one or more nodes happened to be in the plane of section, showed that they take part in the inflammatory process through a marked proliferation of their lymphoid elements. The more acute the course of the disease, the more prominent is the hyperæmia.

The *pancreas*, which certainly plays an important part in the pathology of intestinal infections, has up to the present been almost wholly neglected in anatomic research. I found in the literature only a reference by Nobécourt, to the effect that Arraga-Vinos was able to demonstrate in chronic cases a more or less pronounced sclerosis

of this gland with angiopancreatitis, the latter beginning around the duct. Also some of the gland cells appeared less clearly distinct and their nuclei less readily stained.

On the other hand we have a number of articles dealing with the investigation of the *liver* in acute and chronic diseases of nutrition, the results of which have been collected by Terrien and by Nobécourt. They have a special importance because insufficiency of the oxidizing function of this organ has been repeatedly pointed out as an important factor in the origin especially of chronic disturbances of metabolism. Terrien, to whom we are indebted for the most thorough histologic studies, describes as follows the chronologic development of this process, of which the first stages belong to the acute, and the later stages to the chronic disorders of nutrition. It begins with capillary congestion, and intravascular leucocytosis. Then follows inflammation of the walls of the branches of the portal vein, which leads to swelling and casting off of epithelium, and a beginning parenchymatous degeneration of the liver cells. The most advanced stages of this process are characterized by increase of the above-described lesions, round-celled infiltration of the hepatic tissue occurring in small areas, in places beginning sclerosis and new-growth of bile vessels and at the same time very advanced degeneration of the hepatic cells. Similar observations have been reported by Lesné and Merklen. They give ground for suspicion that many cirrhoses of the liver in later childhood originated in processes of this kind.

For some years the attention of writers has been attracted toward the *kidney* lesions, all the more as the participation of these organs in the disease process is often very significant clinically and occupies the foreground in the symptomatology.

Here also the severity and duration of the disease plays an important part, and on this account the works of different writers show pronounced differences. The epithelium of the convoluted tubules is a site of predilection for the lesions. Kjellberg found there fatty degeneration, while the lumina were filled with fat and granular masses, and the cells of the straight tubules showed cloudy swelling. This finding has been frequently confirmed, and seems to be fairly constant in acute diseases of nutrition of severe course. I have been able to demonstrate it in the majority of the cases examined. The literature on the subject has been collected by J. Pick. He was able to show in osmic acid preparations, that the fatty degeneration was not confined to the places mentioned, but was also found in some glomeruli, and in the epithelium of the Malpighian tufts. In addition, there is marked hyperæmia of the entire renal cortex, proliferation of epithelium in some of the Bowman's capsules, and areas of cell infiltration at the boundary of medulla and cortex. In chronic cases the changes are

mainly found in the vessels, in the form of capillary inflammation, arteritis, phlebitis, and areas of infiltration with leucocytes. Glomerular nephritis also is at times observed (Heubner, de Rothschild, et al.).

The frequent occurrence of spasm, contractures, and paralyses in the course of acute and chronic diseases of nutrition has directed attention to the histologic examination of the *central nervous system*. Zappert believed he had found the anatomic basis of the spastic condition of the extremities so frequently present in a degeneration of the anterior nerve-roots. Thiemich disputes this, failing to find a corresponding lesion in paralysis of the cranial nerves. Also Müller and Manicatide, working with Nissl's method, were able to demonstrate various cell changes, pointing toward an infectious or toxic origin, but were not able to establish a type characteristic of diseases of nutrition.

V. METHODS OF CLINICAL DIAGNOSIS

There are a number of methods used in the clinical diagnosis of the different forms of diseases of nutrition, of a general character. Therefore we will take up these before speaking of the individual types of disease which we have set forth above. Of course only such methods are meant as can be employed without complicated apparatus, and without too much loss of time, and the use of which will be of actual assistance in the diagnosis of the diseases of nutrition. These methods are to some extent those in general use in the practice of medicine; to some extent they are methods modified to correspond with the conditions of early childhood, and to the least extent are they adapted only to diseases of this period of life.

The regular *observation of the body weight* is one of the most important and valuable means of assistance in judging the course and severity of diseases of nutrition, as well as the results of the therapeutic measures employed. Weighing should be done every day in acute cases, at the same hour, and in chronic cases at least twice a week.

The above precedure is a certain standard of measurement. Less certain are the various methods of *milk examination* in use clinically. Certain gross faults of breast-milk can be diagnosed macroscopically. A watery appearance of the milk, or the fact that the drops pressed out of the breast during or after nursing are thin, almost transparent, and do not adhere to the nipple, point to deficiency in fat and other constituents (Epstein). A diffuse light yellow color, or the appearance of yellowish streaks in the expressed milk drops render probable an admixture of colostrum.

The microscopic *examination of breast-milk*, which was extensively employed by Fleischmann, and recently highly recommended by Friedmann, is only of value in so far as it verifies the presence of colostrum corpuscles or pus cells, which the macroscopic examination has

already made probable (Biedert-Winter, Epstein). The estimation of the number of fat globules in the field of view, and their relative sizes has little value. At most, preponderance of the smallest fat globules is useful as a sign of a poor breast-milk.

Also the different lactoscopes, of which there are a great many of different construction, are only useful in recognizing the grossest bad qualities of breast-milk, and accomplish no more than the naked eye.

Umikoff's reaction for testing the age of milk in the sense of the duration of lactation in the nurse, is of little value. It consists in adding to 5 c.c. of milk 2.5 c.c. of a 10 per cent, solution of ammonia, and awaiting the appearance of a rose color. . It is not reliable in practice, as Brudzinski, who found it in the 12th and 14th months of lactation, has shown.

The same is true of Storch's reaction, which depends on the fact that raw milk breaks up hydrogen peroxide into water and free oxygen, the latter being recognized by the appearance of a blue color on the addition of paraphenylendiamin. Thiemich, by thorough researches, has disproved the supposition of Nordheim, that this method is of value in explaining certain cases of failure of a child to thrive at its mother's breast.

Thus we cannot rely upon any of the methods mentioned above, and will not assign to them any important rôle among diagnostic criteria.

It is the same with the simple clinical methods of *testing cow's milk*. One of the most recent is the examination of market milk in cover-glass preparations, advanced by Petruschky and Kriebel. Its value has been repeatedly confirmed (Rabinowitsch, Beck, Piorkowski). The procedure consists in drying a drop of milk on a slide, fixation in the flame, removal of the fat with ether, and staining by Gram's method, and its simplicity permits its wide use.

Examination with the stomach tube, which Epstein introduced into pediatrics, teaches us about a number of deviations from the normal course of gastric digestion. The procedure, of which we will take up the details in speaking of therapeutics, is extremely simple. It enables us in the first place to judge of the motility of the stomach. Normally the stomach should be found empty $1\frac{1}{2}$ to 2 hours after the taking of food, in naturally nourished infants, and at most 3 hours after in the artificially nourished (Epstein, Cassel, Szydlowski, et al.). Every deviation from the normal signifies a diminution in the gastric motility. In the second place the use of the tube makes possible the recognition of mucus, the testing of the reaction of the gastric juice, the macroscopic and microscopic examination of the gastric contents, the chemical tests for the presence of free hydrochloric acid, and organic acids (lactic, butyric and acetic acids), and the microscopic and cultural examination of bacteria. Thus it gives us a valuable diagnostic and

therapeutic finger-post (Bauer-Deutsch, Finizio, von Hecker, A. H. Mayer, Wachenheim, et al.). The methods of obtaining these data do not differ from those in general use.

On the other hand the *examination of the intestinal discharges* plays a much more important part in the diseases of nutrition of infants, than in older children, and also, on account of its technique, it is simpler and better adapted to general use.

Raudnitz, who has done thorough work on this subject, has devised a very convenient reagent-case, which I have found very useful in a form somewhat modified to conform to modern progress. This contains in drop bottles with ground glass stoppers, the following reagents: (1) Distilled water, for moistening the preparations. (2) A 5 per cent. solution of acetic acid, which dissolves triple phosphate crystals and calcium carbonate with gas formation, dissolves Charcot-Leiden crystals and fatty crystals, but does not dissolve oxalate. It also makes the intestinal mucus stringy, and causes the nuclei of leucocytes, epithelium, and so forth to stand out more clearly. (3) A 20 per cent. sodium hydrate solution, which dissolves the fatty acid needles with the formation of soap, and clears up the albuminous substances. (4) 95 per cent. alcohol, which dissolves the fatty soap, partially dissolves the free fats and fatty acid needles, and is used in making the staining and decolorizing fluids. (5) Ether, which dissolves fats, fatty acids, cholesterin crystals, and free bile-pigments. (6) 5 per cent. sulphuric acid, which shows the presence of calcium through the formation of calcium sulphate crystals, dissolves calcium oxalate, destroys fatty soaps, and stains the cholesterin crystals a violet-red. (7) Fuming nitric acid, for Gmelin's test for bilirubin and its salts. (8) Lugol's solution (iodine 1. potassium iodide 2. distilled water 300.), for the Weigert-Escherich stain, and for coloring starch and iodophilic bacteria, and also cellulose; the former become blue, the latter becomes yellowish brown. (9) A filtered concentrated aqueous solution of methylene blue, for staining bacteria and cell-nuclei. (10) A 2.5 per cent. solution of gentian violet in water, boiled for half an hour and filtered; for Escherich's modification of Weigert's staining method. (11) A mixture of two parts absolute alcohol and three parts aniline oil. (12) Aniline and xylol in equal parts. (13) Pure xylol, all three for the Escherich-Weigert stain. (14) Concentrated alcoholic solution of fuchsin diluted one half with absolute alcohol, as a contrast stain in the Escherich-Weigert method. (15) Alcoholic tincture of alcanna, which stains fat red.

By means of these reagents, a complete examination of fresh stools for the various food ingredients and the residue of digestion, can be carried out. The modification of Weigert's staining method recommended by Escherich is used for bacteria. It is performed in the following way:–

PLATE 38.

I. and II. Well-digested breast-milk stool.
III. Discolored stool from a well-nourished breast-fed infant.
IV. Stool with undigested fat and fatty acid particles from a well-nourished breast-fed infant.

(Photographed direct from nature.)

The specimen is spread on the slide, dried, and fixed in the usual way, and is then flooded with a mixture consisting of the gentian violet solution $8\frac{1}{2}$ parts, and the alcoholic aniline solution $1\frac{1}{2}$ parts. This is allowed to remain 2 seconds, and then absorbed with filter paper. Next the iodine and iodide of potash solution is dropped on for a moment, and at once absorbed, after which the aniline-xylol solution is dropped on continuously until no more stain comes off the slide. Then xylol is applied, and finally, the fuchsin solution is allowed to run over the slide for an instant, and is freely washed off with water. The slide is then dried, and examined, either directly, or after the application of a cover-glass. I can most highly recommend this process, which offers a very significant picture in the study of the bacteria of the stools. It is especially adapted to tracing the behavior of the intestinal flora under the influence of various diatetic and therapeutic measures.

In the cultural examination much depends upon the selection of a fresh specimen. Either a sterilized lead tube (Escherich), or a Nélaton catheter, in the opening of which sufficient material collects, (Epstein), may be inserted into the anus, or else the freshly passed feces may be taken up with sterile gauze (Flexner-Holt). Other organisms are so easily overgrown by the exuberant development of the bacterium coli communis on the ordinary culture media, that a marked dilution of the specimen to be examined by the use of numerous plate cultures is desirable. Besides the usual methods, anaërobic cultivation should always be employed.

The reaction can be tested in fresh stools, by means of previously moistened strips of litmus paper.

The ash-content can be approximately estimated by the ignition on a platinum beaker of small particles of the feces (Heubner).

The chemical examination of the stools, in regard to which the odor gives much essential information (Selter), is performed clinically in the following way. The stool is tested for lactic acid,* by extracting with ether, filtering, evaporating the ether, dissolving the residue in water, and adding one or two drops of a solution of ferric chloride in carbolic acid (10 c.c. 1 per cent. carbolic acid plus 1 to 3 drops of ferric chloride). A yellow or yellowish green color appears. The test for acetic and succinic acid, which are further fermentation products of the sugar in the food, is made in the following way. The stool is extracted with water, filtered, and then heated with a couple of drops of alcohol and sulphuric acid, upon which a clear odor of vinegar appears.

By means of the methods briefly sketched above, in connection with the inspection of the discharges, the appearances of which under pathologic conditions will be considered in speaking of the different

* Uffelmann's reaction, which Czerny-Keller ("Des Kindes Ernährung") consider unreliable.

disease types, sufficient material is found for clinical requirement, and for diagnosing the nature of the disturbance as well as the eventual result of therapeutics.

Just as important is the *examination of the urine*, for the collection of which can be used with boys the urine collector constructed by Raudnitz, with girls a metal catheter. Beside the usual albumin and sugar tests, the test for indican must not be neglected. According to Combe its amount is a measure of the degree of intestinal putrefaction, a view of the accuracy of which Lesné and Merklen doubt on the basis of their own researches. The testing of the alimentary glycosuria, and of the elimination of methylene blue has hitherto given no results of practical value (Lensé-Merklen).

Obviously in addition to the chemical examination, there must be a thorough microscopic search of the sedimented or centrifugalized urine, and finally also the taking of cultures.

The test of the toxicity of the urine proposed by Bouchard has proved worthless, as Brieger has been able to show, that if the urine is simply diluted until isotonic with the blood serum of the individual, its toxic action is entirely removed (Combe).

Also the results of cryoscopy of the urine (Nobécourt, Lesné-Merklen), and of the testing of the velocity of its flow with the stalagmometer (Amann) have made but little progress, and are superfluous for our purpose.

The *examination of the blood* can be made from cover-glass preparations stained in the usual way, with counting of the different forms of leucocytes by means of a movable stage. This informs us as to the existence of a leucocytosis and as to its character. However, the reports respecting this in the literature (Japha, Mason-Knox, Wacfield, Zahorsky), sound rather contradictory, so that the prognostic value of such findings must be taken with caution.

The counting of the leucocytes by means of the Thoma-Zeiss apparatus permits an exact estimation of their increase, and of the influence of digestion and the effect of nourishment with cow's milk upon leucocytosis, etc. (Moro).

Also the estimation of the specific gravity of the blood by means of the pyknometric method is, according to Schlesinger, of prognostic value.

The usual method of examining the blood of infants bacteriologically, by pricking with a needle and inoculating on various culture media (Czerny-Moser, A. Baginsky, O. Heubner, Escherich, Pierracini, and Nencioni, et al.), has shown such divergent results, and is beset by so many sources of error, that it had much better not be employed. The only reliable method is the withdrawal of a greater quantity of blood (at least 1 c.c.) by means of a syringe from a vein of the fore-

arm. This proceeding can only occasionally be decided on, because at times the withdrawal of so much blood from a poorly nourished infant can not be undertaken without hesitation, and because at other times it is impossible on account of the small size of the veins and the peripheral anæmia of the body. Lesné in one case has had recourse to puncture of the longitudinal sinus through the greater fontanelle, a procedure which should not find many imitators.

The agglutination test, a proceeding easily carried out clinically, has brought about no practically useful results in respect to the bacterium coli, but has been instrumental in shattering the supposed position of this organism in the pathogenesis of gastro-enteric affections (Nobécourt, Escherich, Pfaundler, Widal, Lesage, Templier, et al.). It appears to give more constant and more clinically valuable results with the different types of dysentery bacilli (Flexner-Holt, Jehle).

There remains *lumbar puncture* to be mentioned here, which can be employed in cases where nervous symptoms accompany diseases of nutrition. It is used as a diagnostic procedure in differentiating between the functional nature of such symptoms, and meningeal or cerebral complications (Nobécourt).

VI. DISTURBANCES OF NUTRITION IN BREAST-FED INFANTS

A. **From Overfeeding.**—This form represents the commonest type in exclusive breast-feeding. As may easily be understood, it is met with more frequently, and in a more obstinate form, in infants nursed by a wet-nurse, than in those nursed by their own mother. It is more often present in the first months of life than in older infants on account of the slight development of the function of the digestive organs in the former, and because in many cases a reciprocal adjustment occurs which leads to spontaneous cure.

The normal course of gastric and intestinal digestion requires that the time necessary for the emptying of the stomach be maintained. This time is necessary for the production of free hydrochloric acid by the gastric glands, which product on the one hand sets a definite limit to the growth of bacteria in this food reservoir, and on the other hand, as we know from the researches of Pawlow and his pupils, acts as a stimulant to the secretion of bile and the pancreatic juice. Too frequent feeding at the breast causes injury, because the stomach, not yet fully evacuated, is filled with a fresh supply of food, which seizes upon the quantity of free hydrochloric acid already prepared, combines with it, and thus prevents its stimulant action on intestinal digestion. Thus a vicious circle is produced, because through the intimate physiologic connection between gastric and intestinal digestion every injury affecting the one soon manifests its effects upon the other. Even when the intervals are chosen with proper regard to the time necessary

for gastric digestion, the same results may be caused by offering too great quantities of breast-milk. In this case, in addition to the taking up of the free hydrochloric acid, a further factor appears in the overdistention of the stomach. This leads to injury to the motility of the stomach, which in turn reacts upon the secretion of the gastric glands, for in this little chemical household all the factors are linked with one another. When both of these evil conditions are combined, too frequent feeding, and too much at one time, it is very evident that the disturbances will be all the more easily produced, and will run a severer course.

There are a number of physiologic aids, by means of which the infant tries to ward off these injuries, and which appear as the first symptoms of overfeeding to be perceived clinically. First to be mentioned is the *regurgitation* of the food. The stomach, overburdened by too abundant or too frequent meals, gives back a part of the food immediately after taking it, and the milk, unchanged in character, pours out of the mouth immediately after or during nursing, without any visible straining or previous discomfort. This symptom is met with in children whose appearance and increase in body weight are entirely favorable, which must be the origin of the old saying,—"spitting child—thriving child." If no attention is paid to this symptom, as is usually the case on account of the environment of the infant, then *diarrhœa* appears as a further symptom of defence. Instead of the normal two to three movements, there are four to five in twenty-four hours, and their nature points on the one hand to overloading of the bowel, and on the other hand to insufficient utilization of the food resulting from its accelerated passage through the intestine. Finally a *lessened appetite* is associated as a third attempt at natural protection. The child constantly applied to the breast always seizes the nipple well, but lets it go again after a few short sucks, or, if the feeding intervals are regular, it takes less food each time.

Unfortunately these measures of protection are deceptive in character, and their significance is usually misunderstood, because their progress is always apparently favorable, even if not uniform but rather by fits and starts, and soon oversteps normal limits. Consequently they are insufficient to enforce upon those caring for the child a proper understanding of their significance, and thus the evil goes unceasingly forward, and soon the boundary line of the pathologic is passed over. When this occurs the various symptoms become more marked and more constant. Whereas at first the regurgitation of the gastric contents only occurred occasionally, and always consisted of unaltered milk, it now follows every feeding, and there soon occurs a postponement of its time of occurrence, so that the giving back of the food no longer follows immediately upon its taking, but occurs after a longer

interval, from some minutes to half an hour. Moreover the regurgitation is accompanied by visible discomfort, and produces an altered milk, which contains larger or smaller curds, as well as lumps of mucus, floating in a grayish white serum, and is sometimes colored yellow from admixture of bile which has gained access to the stomach. The vomitus has a strongly sour, and sometimes pungent odor. An equally sour belching frequently occurs in the intervals between the vomiting and causes the infant pain, which manifests itself in discomfort as betrayed by the features, so that instead of the lack of expression of the first weeks of life, its appearance assumes the more or less clear characteristics of suffering. The examination of the gastric contents in this stage shows in the first place a notable slowing of the gastric motility, as, instead of the normal empty condition after two hours, considerable altered contents are found in the third or fourth hour. The free hydrochloric acid is mainly or wholly lacking, while on the other hand the acids of fermentation, lactic, butyric, and acetic, are found present. The number of microörganisms has become greater, and their varieties more numerous.

The intestine reacts to the lessened stimulus from a smaller or absent hydrochloric acid production, at first mainly by a diminution of its activity, and constipation. Later the pathologically altered gastric contents enter into the intestinal canal, together with the abnormal products of acid fermentation which also alter the conditions of growth of the intestinal flora. All this causes increased gas production and increased peristalsis. Instead of the normal movement, that golden-yellow, glistening, homogeneous, mushy product with its agreeably sour odor, which two or three times a day pours out of the anus like syrup out of a bottle and is first discovered on undressing the child, the process of defecation now assumes a much more dramatic character. It is ushered in by restlessness, the entire body starts into activity, growlings and gurglings are heard, and finally, with a loud passage of gas and a vigorous cry, a fluid mass comes out of the rectum as if squirted out of a syringe. This mass on inspection of the napkin appears to be surrounded by a fairly wide watery border, and shows larger and smaller white fragments, which here and there are light or dark green, and from it there rises a disagreeable, sour, pungent odor. The test of the reaction gives a higher degree of acidity than is found in the normal movement, Gmelin's test shows abundant bile-pigments, the microscopic examination shows rare fat drops and numerous fat needles and fat clusters (crystallized fatty acids), and the bacteriologic picture shows little deviation from the normal, and a preponderance of thin Gram-staining rods. Also, green stools may be produced from the very beginning, alternating with the curdy form, or else on standing a greenish coloring of the movement beginning at its outer border may

come on, appearances the causes of which are not yet entirely clear.* The entire nature of the child is changed. The alteration in its expression, which has already been mentioned, soon not only accompanies the nausea preceding vomiting, or the pain attending evacuation, but becomes permanent as a result of the persistent intestinal discomfort. Epstein, a fine observer of such things, gives the following description. "The appearance of content disappears, even in sleep, the features become more shrivelled and more pointed, the glance becomes weaker, the corners of the mouth more drawn back, and the expression of the face becomes more set and pinched."

Sleep, which normally comes on after feeding with evident comfort, either fails to appear at all, or lacks its normal depth and quiet. The slightest noise makes the child wide awake, and even without such disturbances it suddenly starts convulsively, works its lips, arms and legs in an unaccustomed way, in short, does not obtain proper rest. The face has become notably paler, the rosy tint of the skin which after the hyperæmia of the first few days appears as a sign of health, being replaced by a slight anæmia. Macules and papules of light or dark red color appear on the face, either singly, in groups, or as a diffuse erythema. The genital region is frequently the seat of processes varying from simple reddening of the skin to hard infiltrated and superficially ulcerated or even deeper eczema, which can extend down to the knees, or up to the middle of the back. The abdomen, which in normal infants is scarcely distended, and does not reach above the level of the thorax, now appears distended by gas, with tense skin. On applying the hand to the abdomen, the movement of the intestines is felt, and occasionally, with a loud noise, sour-smelling or foul gas comes out of the anus, and is frequently accompanied by little spurts of feces. The discomfort of the waking or sleeping infant is increased by attacks of colic, which cause severe crying, in which the face becomes dark red, and the features distorted with pain. The legs are drawn up, or kicked back and forth continuously, thus causing reddening or excoriation of the heels. These symptoms continue until the passage of gas, or a movement of the bowels, which squirts out for some distance, procures quiet for a time.

The mouth, which in the beginning of the symptoms presents a slight reddening, shows in their further course a catarrhal swelling. The tongue shows a velvety coat, which becomes thicker near the base. In very young children a growth of thrush quickly appears which covers the inner surface of the lips, the gums, the mucous membrane of the cheeks, and the palate and pharynx with larger or smaller, isolated or confluent, deposits of a white or yellowish color, which stand out sharply

*They have recently been attributed by Wernstedt to the action of an oxidizing agent found in the intestinal mucus, which is said to cause an alteration in the color of the bilirubin.

from the reddened and somewhat swollen mucosa. There is usually no fever, at most a brief initial rise of temperature.

As to the weight curve, in the beginning it shows a more interrupted course instead of the normal steady increase. Periods of stationary weight alternate with occasional sudden increase beyond the normal rate of growth. Later the weight curve becomes level, or slowly inclines downward. This becomes manifest in the appearance of the child. Its superficial fat and the musculature feel more flabby, its movements lose their vigor, and the skin loses its color.

I have already said that these symptoms of overfeeding frequently subside of themselves, because on the one hand a strengthening of the child's digestive power comes on, which makes it sufficiently developed to meet the increased demands, and because, on the other hand, a regulation of the amount of milk provided by the mother or nurse occurs, which limits the quantity of food given. But one must not count too much upon this spontaneous subsidence, because, although these disturbances are innocent as a whole, and quickly and easily allayed by proper treatment, still the boundary lines which separate them from the severer affections of the gastro-enteric tract are shifting. Furthermore there is the danger of secondary infection, against which the normal course of digestion guarantees a relative immunity, but to which children thus injured by overfeeding are easily liable.

The **treatment** of this condition consists of three parts, prophylactic, causal, and symptomatic, and although these three efforts, as may easily be seen, are interlocked one with another, we will speak of them separately.

The **prophylaxis** accords with our modern point of view as to the appropriate natural feeding of the infant. It avoids giving any food on the first day of life, except perhaps weak tea to boiled water, and from the second day accustoms the child to $3\frac{1}{4}$ to 4 hour intervals between feedings, with an interval of 5 to 6 hours sleep at night. The quantitative conditions are regulated by the nursing mother herself, because the stimulus exercised by the child upon the breast in sucking causes a transition from the stage of colostrum secretion to that of milk production. The quantity prepared, if a normal digestive function be assumed, is exactly fitted to the requirements, while the sucking efforts rock the child to sleep, from which, as the observations of Czerny show, it wakes after three or four hours to the need of taking more food. Meanwhile the transition of the movements from meconium to the normal milk stools is normally completed. Under these conditions, the undisturbed quiet of the house, the rosy color of the child, its lively and vigorous condition when awake, its looking around and active kicking, the firmness of its flesh, and its regular gain in weight, are certain evidence that we are on the right road toward a thriving

growth. With judicious directions on the part of the physician and their proper comprehension on the part of the mother, although indeed the directions frequently have to be carried out only after a tiresome conflict against the various influences ruling in the nursery, everything goes well, and, with a few exceptions still to be spoken of, the dangers of overfeeding are avoided.

It is more difficult with children fed by a wet-nurse, who is provided too soon with a full dinner-pail, instead of having to earn her living by her own work, and all the more as rustic breasts usually flow very abundantly, and every physician takes a certain pride in seeking out as especially desirable a wet-nurse with a lot of milk. Consequently a holding back is called for in the first few days, and the child should be only infrequently nursed. Consequently it is of advantage for the wet-nurse to bring with her her own offspring, to drink up the superfluous milk. Also if the normal number of breast-feedings has been attained, as is usually the case by the eighth or tenth day, the amount taken at each feeding can be controlled by weighing. By comparing this with the average amount the child should take daily (according to Marfan, 15 per cent. of its body weight, according to de Rothschild 125 grams per kilo), one can judge if the proper amount is being much exceeded. If this is the case, the intervals are lengthened, or if this cannot be done because the longest intervals are already being observed, the amount taken at each nursing is reduced, by leaving the child at the breast for a shorter time, or, if the flow is especially abundant, by partially emptying the breast beforehand with the breast-pump. The opinion that every cry of the infant is synonymous with hunger is easily combated in the mother by arguments based on reason, but in the wet-nurse must be especially strongly counteracted, and all the people around the child must be instructed to watchfulness in this direction. Except in severe disease, the breast is always taken eagerly. It will frequently happen that the nurse will give the breast to a crying child sooner than undress it to convince herself that it is not lying in a wet or soiled napkin, or that the clothes are not pressing on it, or something similar.

Just as we have few really efficient measures beside the sucking reflex of the child, to increase the activity of an insufficiently secreting breast, we can as easily dispose of the medicinal and dietetic measures directed to the opposite result. If the nurse were made to suffer thirst in order to reduce the quantity of her milk, it would result in failure, because it is an utterly useless torture, and also the influence of diet upon the quality of the milk has been much exaggerated.

If the prophylactic measures have not proved efficient, or, as is more often the case, if we are confronted with the condition of overfeeding already fully developed, then the second division of the treatment

the dietetic treatment, finds its place. Its fundamental principles are: first, emptying the stomach and bowels of food altered by abnormal fermentative processes, and second, resting of these overexerted organs. This is best done by giving a bland diet for 12 or 24 hours or perhaps even longer according to the duration and severity of the symptoms. This diet consists of boiled water, in case of necessity sweetened with saccharin, or of very weak tea, or, if absolutely necessary in order to not resist the wishes of the family, fennel or chamomile tea. Thus I avoid all other treatment and wait to see whether under the influence of this proceding the vomiting stops, the movements become less in frequency and amount, the passing of gas ceases or becomes much less, and the child becomes quiet. If this is the case, as it is in a large percentage, then the breast is gradually resumed, and, on the day following the period of absolute starvation, may be given twice within 24 hours. Then, if there is no return of the symptoms calling for a repetition of the water diet, it can be given gradually more often until 4 or $3\frac{1}{2}$ hour intervals are attained. If the period of withholding food does not lead to a cessation of the vomiting, I next resort to *washing out the stomach.*

This procedure was introduced into the therapeutics of infancy by Epstein, in 1880. Kussmaul's instrument, reduced to correspond with the smaller anatomical conditions, is employed. It consists of a funnel holding about 100 Gm. ($3\frac{1}{2}$ oz.), and attached to this a rubber tube from $\frac{3}{4}$ to 1 metre (2–3 ft.) in length. A glass tube about the length of the little finger serving as a window connects the rubber tube with a Nélaton catheter (sizes 9–22, Charriére's scale), which has an opening in the side. The catheters opening at the end are condemned by Epstein on account of the danger of wounding, and I myself do not like them, because the relatively small opening is easily obstructed, thus delaying the operation. The child, rolled up in its pillow which confines its arms, is either laid on its back or held upright; I prefer the latter position, because it almost entirely does away with the possibility of an overflow of the fluid into the air-passages. The catheter is first soaked in lukewarm boiled water, or perhaps in Heubner's physiologic salt solution. Water is allowed to run through funnel and tube in order to expel the air. Then the catheter is introduced along the posterior wall of the pharynx, while the free hand depresses the tongue. It glides down without the least difficulty, and when the fundus of the stomach is reached, the funnel is depressed in order to allow the gastric contents to flow out into a vessel placed near by. The funnel being still held down, is filled with the wash fluid at the body temperature, and is then raised up and held till the fluid has all flowed in, when it is again depressed and the fluid allowed to run out. This is repeated until the wash water comes out entirely clear. I have never encountered disagreeable occurrences in the numberless stomach washings which I

have performed in the course of years (an older child reacted violently to the introduction of the catheter, though without further bad results). Consequently I must designate this procedure, especially in the early months with their slight reflex excitability, as a method without danger, and easily practiced. Washing is best performed some hours after the last feeding, and care should be taken, that in the next few hours neither food nor drink be given, as this excites vomiting.

This procedure accomplishes the removal of fermenting material, the washing clean of the mucus covered gastric mucosa, and excitation of the secretion of its glands through the introduction of the tube. Beside this, it exercises a certain influence upon intestinal peristalsis. It is not always sufficient to perform it once, for, especially in cases of longer duration in which the existence of gastric dilatation is assumed, it is necessary to repeat it two or three times. The use of special washing fluids, or the pouring in after the end of the washing of medicated solutions is at least superfluous, and now hardly ever practiced.

The emptying of the bowels, if it has not been attained through the water diet, can be accomplished with mechanical or medicinal means. A useful measure is *irrigation,* for which an instrument is used which consists of a Nélaton catheter of proper size, connected with a hard-rubber stop-cock, to which is attached a rubber tube about one metre in length, and a graduated glass irrigator holding half a litre.

Other apparatuses, involving the introduction of solid instruments into the rectum, are of little value, and dangerous. The soft instrument, smeared with a clean oil, must be pushed up rather far, as otherwise the internal sphincter will form an obstruction to the outflow of the water. Too high a pressure must not be used (about two feet). The child should be laid on the side, with its legs drawn up against the abdomen, and a pillow should be put under the hips to raise them. If the outflow does not occur at once, or if it stops, it can be started up again by twisting the tube, or by pushing it up and down. Boiled water at body temperature is all that is needed for the irrigating fluid, as the form of disease under discussion offers no indication for medicated irrigations. It is usually sufficient to let half a litre of water flow through, and only in older children (over four or five mouths) is a greater quantity necessary.

When the combination of a water diet with irrigation does not suffice to empty the intestine of the accumulated fermenting masses, we have recourse to *purgatives.* Of these calomel has for a long time been the favorite, because, besides having laxative power, it has been considered an antiseptic, and to have an action increasing the flow of bile and the secretion of the intestinal wall. Not only is there little foundation for this view, but through Tissier's researches we have learned of an undesirable effect of the drug, consisting in pathologic

alteration of the intestinal flora. This has strengthened me in my aversion to calomel, although I have never belonged among its supporters. It can be prescribed in purgative doses, .05 gram ($\frac{3}{4}$ grain) up to 3 months, 0.1 gram ($1\frac{1}{2}$ grains) from 3–12 months, divided in two doses. It is insoluble, and must be mixed with some vehicle, or powdered and given in a spoon with some fluid. It can also be given in smaller divided doses, .005 gram ($\frac{1}{12}$ grain) every hour, or .01 gram ($\frac{1}{6}$ grain) every two hours till a total amount of .04–.05 gram ($\frac{3}{4}$ grain) has been taken. In every case one should stop giving it when the characteristic loose leek-green movements have appeared, the color of which is due largely to the formation of sulphide of mercury in the feces (Schoen-Ladniewski).

The same effect can be obtained in an absolutely safe and effective way by the use of other purgatives, of which I can recommend castor oil, in teaspoonful doses, or Hufel's powder (magnesia with rhubarb), as much as can be held on the point of a knife, or Curella's powder, (compound licorice powder) in the same dose, or a mixture of equal parts of hydromel infantum and fluid extract of rhubarb, a teaspoonful every one or two hours till effective. One should stop giving the purgatives when loose stools containing no milk-residue have been established, which usually takes place with an abundant passage of gas.

One of the most tormenting symptoms is the *colic*, which often prevents the child from resting by day or night, and which proves very painful for the family. Usually the colic can be allayed by removal of the cause, through emptying the intestinal canal of its abnormal contents, and quieting it. Frequently however it is necessary, when colic is the prominent symptom, or when it outlasts the other symptoms, to proceed against it directly, in order to procure at least a temporary quieting and thus to allow the infant a few hours' sleep. Often it is sufficient to bring about the passage of intestinal gas or some feces, simply to introduce and withdraw an intestinal sound or empty syringe, or to insert a glycerin suppository. Applications to the abdomen in the form of warm compresses, chamomile bags, flaxseed poultices, or properly formed thermophor plates are also often of temporary service. Also one may employ massage of the abdomen, with the hand moistened in warm oil, grasping the abdomen with the fingers in constantly widening circles from the navel outward, and finally moving along the course of the large intestine, to press out the gases in this way. The internal administration of caraway water (aq. carminativa 30–40 Gm. (one ounce) with aq. destill. 70–60 Gm. (two ounces) a teaspoonful every two hours), or of chamomile and fennel tea is usually but slightly effective. If there are great pain, frequent repetition of the attacks and almost total loss of sleep, we can use chloral, either by mouth or by rectum, as has recently been recommended by Epstein and Czerny-Keller.

Chloral is given in solution, 0.5 Gm. (7½ gr.) to 100 c.c. (3 oz.) of water (one half per cent. solution), one teaspoonful every one or two hours, or half a gram of chloral in 50 c.c. of water (7½ grains in 1½ ounces) can be injected as an enema. As to bromide preparations, I have never seen a noticeable effect. Opium, which is widely recommended (Escherich, Fenwick, Filatow, Jaquet, Soltmann, et al.) can be given either by mouth, one or two drops of tinct. opii simplicis (P. G.) in a 100 gram solution, a teaspoonful every two hours, till quieting occurs or it can be given by rectum, one or two drops of tincture of opium to the enema, not oftener than once a day. It has this drawback, that it has a constipating action, and consequently after its temporary effect it has a tendency toward increasing the attacks of colic. Also we must take into consideration the intolerance toward even the smallest doses of opium which is often found, especially in very young children, and which manifests its effects in severe symptoms of poisoning. Recently, in consideration of the remarkable effect of morphine in intestinal colic, I have ordered it in especially obstinate cases. I give muriate of morphine .001 gram with aq. 100. ($\frac{1}{60}$ grain with 3⅓ ounces of water), and a saccharin tablet, a teaspoonful every two hours till quieting occurs. It has a less marked constipating action, and I have seen a good result without any injurious action.

With marked sour fermentation in the stomach, recognized by the pungent odor of the vomitus and the sour belching, mild alkalies perform good service, such as sodium benzoate, or magnesium benzoate in 3 or 4 per cent. solution, or small doses of sodium bicarbonate, as much as can be held on the point of a small knife, after each feeding. When the stomach washing has shown marked retardation of digestion, one can give some hydrochloric acid and pepsin before each meal. For example, Soltmann's formula can be used: pepsin 1.0 Gm. (15 gr.), acid hydrochlor. gtt. x, aq. destill. 100.0 c.c. (3½ oz.), saccharin tablet, one teaspoonful before each meal.

Lactic acid, recommended by Hayem and Lesage for green diarrhœa, I could not get the children to take in the quantity recommended by these authors (15–20 gram (½ oz.) a day) and saw no results from smaller amounts.

When the diarrhœa is very marked, it can be combated by bismuth preparations (bismuth subnitrate or salicylate in 2–4 per cent. emulsion with mucilage of acacia) or by tanningen, tannalbin, bismutose, etc., but one must not forget to warn the family of the resulting dark color of the movements, otherwise much anxiety may result.

Thrush usually becomes less marked with improvement of the digestive symptoms, and therefore should never form the sole condition requiring treatment. Still, local treatment of thrush is often not unpardonable. For this I prefer a corrosive sublimate solution,

1 to 5000, which is painted on every two hours. Epstein recommends a 5 per cent. sodium borate solution, Escherich recommends the boric acid "comforter," for which proceeding I cannot be enthusiastic, as it restores to honor a nuisance banished with difficulty from the nursery. At all events, in any method of treating thrush every brisk proceeding should be avoided, and the removal of the mycelium should be undertaken in the most careful way, with a good light and under control by the eye.

Intertriginous eczema requires in the first place the greatest attention on the part of those in charge of the child, frequent changing of the diapers, the avoidance of the use of diapers which have become wet and have been simply dried without washing, together with delicate cleansing of the anal region with cotton and vaseline to prevent mechanical injury. The treatment should in the first place be directed to the digestive disturbances, since their alleviation deprives the movements of their power of irritating the skin, and is frequently sufficient in milder processes to bring about spontaneous cure. If this does not occur, local treatment must be tried. In a purely erythematous form glycerite of tannin in 5–10 per cent. solution is best. Strips of cotton are soaked in it, squeezed out, applied, and changed after every movement of urination. Old diapers should be used, as the solution stains the linen. Or a powder may be used, for example acid salicylic .25 Gm. (4 gr.), amyli, zinci oxid. aa 15.0 Gm. (4 dr.). In forms with exudation, the parts are bandaged with lassar paste (acid salicylate 0.1 to 0.2 Gm. (1½–3 gr.) amyli, zinci oxid. aa. 10.0 Gm. (2½ dr.), vaseline 20.0 Gm. (5 dr.) M. ft. pasta), which is smeared on with the clean finger, and then thickly strewn with rice flour. In younger infants, less salicylic acid should be used, on account of its irritative action. When the crusts formed by this paste get cracked, fresh paste and powder are applied. With marked infiltration of the skin poultices are used, with either lead water, or dilute aluminum acetate solution [alum 5.0 Gm. (1 dr.), plumbi acetat. 25.0 Gm. (6 dr.), aq. destill. 1000 Gm. (1 quart)]. Bathing is always to be stopped in intertriginous eczema, because it has been found empirically that the process is thus more quickly cured.

B. **From Insufficient Food.**—This form of disease is much less common than the one just considered. Its cause is insufficiency of the nurse's milk, and its recognition is of importance, because it points to the only efficient method of treatment. Its recognition is however not wholly easy, since the condition is often confused with emaciation due to some other cause.

We have no reliable method of measuring the milk content of the female breast, and are therefore compelled to depend upon the information gained by weighing the child before and after nursing. Often we can convince ourselves that breasts, which are apparently poorly

developed, in which we feel only a little glandular tissue, and which yield on pressure only meagre drops of milk, are nevertheless quite sufficient for the needs of the infant, because it drinks enough and gains weight. On the other hand, an apparently full breast can yield too little food material, on account of overdevelopment of the fatty tissue at the expense of the secreting parenchyma. This condition has been called "hypogalaktie" (Marfan), and primary and secondary "hypogalaktie" have been differentiated, according to whether lactation is insufficient from the outset or whether after a temporarily sufficient production of milk, it begins prematurely to dry up. But care must be taken in making this diagnosis, all the more in the case of nursing by the mother, especially if a primipara, for not infrequently they have difficulty at the beginning, but with sufficient perseverance they are able to nurse successfully.

Perhaps the question may arise of the propriety of placing under the heading of diseases of nutrition, a condition of the child which is simply that of hunger. For my part I believe that to do so is thoroughly justified, because in the first place the disturbances arising as a result of insufficient feeding, if they persist over a certain time, can lead to severe changes in the body, and can make it susceptible to secondary infection of various kinds. And in the second place symptoms appear as a direct consequence of insufficient food which cause relief by making the taking of food difficult or impossible.

A pardonable digression may here be made to the subject of the physiology of nutrition. The newborn infant usually takes on the first day of its life either no food, or very little, and also the amount of colostrum taken before the appearance of regular lactation is very little, so that we reckon regular nutrition as beginning on about the fifth day. This of course holds good only for infants nursed at the mother's breast. Marfan gives a table, showing the average amount of food taken in 24 hours and the quantity at each feeding, which is taken from the observation of healthy and thriving infants. The figures are perhaps somewhat high, and the intervals rather short, but nevertheless they serve as an approximate standard, especially if the intervals are reduced to the three hours customary with us.

Infant's Age.	Number of feedings in 24 hours.	Interval between feedings.	Amount of milk taken at each feeding.	Total amount taken in 24 hours.
1st day	1–2	3 hours	4–5 grams	8–10 grams
2nd day	6	3 hours	8–10 grams	48–60 grams
3rd day	7	3 hours	15–20 grams	105–140 grams
4th day	7	3 hours	20–30 grams	140–210 grams
5th–30th day	7	3 hours	40–75 grams	280–535 grams
2nd and 3rd month	7	3 hours	75–100 grams	535–700 grams
4th and 5th month	7	3 hours	100–120 grams	700–840 grams
6th–9th month	7	3 hours	140–160 grams	980–1120 grams

According to Czerny-Keller the 24 hour amount of food taken by the healthy breast-fed infant in the early weeks is about one-fifth of the body weight. From the middle of the first three months to the middle of the second three months it falls to one-sixth and one-seventh of the body weight. From six months on it remains at one-eighth of the body weight.

In children with healthy digestion nursed by a healthy mother there occurs a constant *gain in the body weight* averaging 25–30 grams a day. Under such conditions, the duration of nursing is from ten to at most twenty minutes. In the early months the child when satisfied falls asleep at the breast, to wake up only for its next feeding, while older children if they do not fall asleep, let go of the nipple with a marked expression of satisfaction, and pass the time till the next feeding in quiet content and serene humor. The movements normally formed and golden yellow occur two to four times a day; the abundant secretion of urine, the rosy tint of the skin, the firmness of the flesh, the abundant subcutaneous fat, and the formation of the typical fatty folds in certain parts of the body, are further signs of thorough nutrition and normal development.

In the insufficiently nourished breast-fed infant, such as drinks enough milk to build up its body substance to some extent, but in which, for instance, the daily gain in weight is only 10–15 grams instead of the average, there is less development of the subcutaneous fat, so that such infants appear thin and lack the plump outlines of the young baby. The abdomen, which under normal conditions shows a rounded outline, is flat, often actually somewhat indrawn, the movements occur at most twice a day, their amount is relatively small, their consistency somewhat harder, their color rather approaches a dark yellow ochre than the golden yellow of the normal milk stool. The passage of urine, which in thoroughly nourished infants occurs 10 to 15 times in 24 hours and which always makes a widespread spot on the napkin, is much restricted in frequency and amount, and the duration of nursing appears prolonged over the normal.

From these cases near the border line, various transitions lead to the more or less complete condition of *inanition*. This is characterized by the fact that the child, after the physiologic loss of the first few days, instead of showing a constant and lasting gain in weight from the end of the first week on, remains at first stationary in weight, or shows up and down oscillations, till finally a slight but constant loss becomes established. He then appears thin, the abdominal wall is notably indrawn (Czerny-Keller), and the fontanelle is slightly depressed. The movements are notably constipated, often only two or three a day, at times occurring only by means of artificial aids, and are dark brown or greenish black in appearance, their sticky consist-

ency reminding one of meconium. The amount of urine is reduced to a minimum so that the child when undressed is almost always found dry. Weighing shows us that the amount taken at a feeding is of small value, and that the total amount of milk taken in 24 hours is far behind the normal figures mentioned above. Also the drinking of such children is very characteristic, although we must convince ourselves that it is not due to some malformation in the buccal or nasal cavities hindering sucking. They lie for a long time at the breast, and make periodical sucking movements, but one can not hear the sound of swallowing which in normal infants occurs after every few sucks and is evidence of the passage into the stomach of the milk collected in the mouth (Tarnier and Chantreuil, cited by Bidoult). Moreover, no drops trickle down from the corners of the mouth, and the expression of the face does not show satisfaction.

Such insufficiently nourished infants are usually not very restless, nor do they pass the time between feedings in crying, but they sleep a great deal on account of their lower vitality, and must be waked up to nurse. They also feel cool, and in this respect, as Budin pointedly remarks, remind one of premature babies. The same author has also made the important observation, that with a longer duration of this condition an inability to swallow comes on, so that nutrition even by means of a spoon is not successful, and must be effected by tube-feeding (gavage).

If the under nutrition is of a slight grade, with constant though slight gain in weight, the stools showing a milk residue, and the passage of urine occurring several times a day, then we can simply wait without hesitation, since experience teaches that such conditions improve with time, because the sucking stimulus constantly exercised by the child causes finally a more abundant secretion of the breast-glands of the mother. Such a result is still more quickly brought about, when a strong and vigorously sucking child is put to the breast of a mother with insufficient milk, a procedure which does very well in hospitals, but in private practice can usually not be carried out. One can seek a similar measure of help by trying several times a day partially to empty the breast by manual expression or by the use of a breast-pump, but these manipulations have but little value.

The causes of this primary "*hypogalaktie*" are not wholly clear, though indeed in very rare cases it may be traced to an under development of the breast-glands resulting from a possible hereditary or racial influence. Besides this primary "hypogalaktie", which in its mildest form presents itself as a belated appearance and slow establishment of lactation, there is a secondary form, which can come on at various times in the course of a lactation hitherto favorable. It is often in a certain sense a physiologic process, in that glands which have for a long time

functioned properly, simply cease to secrete, which gives a natural appearance to the failure of lactation. This finds its clinical expression in the child, in a stand-still in weight, and beginning constipation; and in the mother by the breasts becoming emptier and cooler. To be differently interpreted are those cases in which a failure of secretion occurs after a relatively short duration of activity, without any excessive demands (as perhaps too frequent nursing), having been made upon the breast. Such a condition is shown by weighing the infant before and after nursing, and thus finding the amount of milk drunk insufficient. It is often temporarily observed during menstruation, when we must simply wait to see whether or not it persists. It can become persistent through the supervention of pregnancy. In wet-nurses, the long journey to the place of their engagement and the changed conditions of life in their new sphere of activity frequently bring about a similar temporary diminution in their milk; but this usually lasts but a short time. On the other hand, nursing mothers of the upper classes who are often of inferior physical strength, frequently show at first a sufficient or abundant supply of milk, and later a relatively early diminution and premature disappearance. Nevertheless, the influences of diet, and of psychical affections, whether sudden or lasting in their action, have been much exaggerated. The former, if it does not directly affect the health of the nurse and produce disturbances of the appetite and digestion, is without any importance. Psychical factors might affect the quality of the milk (although this also is not extensively proven), but they are without effect on the quantity.

The next question is, *What mode of treatment should be adopted when the breast-milk is undoubtedly insufficient?* Once more it must be emphasized that in the first place the existence of this condition must be indubitably established. Other processes leading to cachexia must be excluded, such as tuberculosis, syphilis, chronic diseases of nutrition which usually cause diarrhœa and not constipation, malformation of the anus, rectum, or other parts of the intestine interfering with the expulsion of the feces, and affections of the nose or mouth causing difficulty in sucking. It is further to be noted that the thorough understanding of this particular condition requires a rather long observation of mother and child, at least 10 to 14 days, and that it must not be forgotten that early deficiency, especially in primiparæ, frequently improves spontaneously, and consequently a retarded lactation should not make the physician impatient.

After careful weighing of all these circumstances, we can consider the treatment. This is effective only because the most important and for the present the only really valuable therapeutic aid, namely, the stimulus excited by the child in sucking, brings about the desired result.

To accomplish this most effectively, if there is not a second child

at our disposal, we put the infant to both breasts each time, and perhaps let it nurse more often. If we do not obtain results after a trial of two or three weeks, we resort to mixed feeding, the "allaitement mixte" of the French authors. This can be carried out in two ways (Budin). By the first plan, we ascertain the difference between the daily amount of breast-milk taken and the amount which should be taken. Then we may use bottle feedings of cow's milk, sterilized and properly diluted alternating with the breast, the amount given at a feeding being so adjusted that the total number of feedings will make up the deficiency. By the second plan we may add to each breast-feeding the amount of nutriment which is lacking in the form of cow's milk. The first method has the drawback, that the children are not put to the breast often enough, and also that they soon prefer the easily sucked bottle, and either refuse the breast entirely, or else do not suck with the force necessary to increase the secretion. For these reasons the second method is to be preferred, and by its use it not infrequently happens, that in the course of a few weeks the secretion of the maternal breast has been so much increased, that the additional feeding can be steadily reduced in amount, and finally entirely abandoned.

There are numerous measures which have been recommended to increase the secretion of the mammary glands. Their very multiplicity gives rise to a doubt as to their value, a doubt which is confirmed by practical observation. I will only remark very briefly that neither from somatose, from Heyden's "nährstoff," nor from laktagol have I seen any result, and I have employed faradization of the breasts without effect. As to the treatment proposed by Bouchacourt (cited by Marfan) consisting in the administration of sheep's placenta, for which a theoretical basis at least cannot be denied, I have had no personal experience. The giving of large amounts of milk to the mother is often resisted and is of no value. The various preparations of galega officinalis (Marfan) and numerous other measures should, as Marfan says, be used only to meet the eagerness for therapeutic accomplishment on the part of the physician, and to attain results on the part of the mother by suggestion. In so far as they are of a harmless nature they can be employed now and then. If the measures mentioned above do not in a relatively short time accomplish the desired result, one is compelled in order to meet the danger of loss of the power of swallowing, and fall of body temperature, to put the child to a freely flowing breast, since under such conditions artificial feeding has few chances of success.

As to a hired wet-nurse with primary hypogalaktie, such a nurse should not be accepted. If a temporary diminution of the milk supply comes on as a result of change of diet, the appearance of menstruation, or similar causes, one simply waits quietly for the supply to increase again. If the diminution persists, another nurse is secured.

If we have to do with secondary hypogalaktie, our procedure is also variable, according to whether we are dealing with the nursing mother or a hired nurse. In the former case, we first try mixed feeding. If this does not produce the desired result, or if the child is still young, under the fourth or fifth month, we will engage a wet-nurse; if it is older, one can wean it. Secondary hypogalaktie of a persistent nature in a wet-nurse with a young child calls for a change of nurse; in an older child, beyond the first six months, it calls for weaning.

C. **From Unsuitability of a Special Breast-milk.**—Cases belonging to this category are relatively infrequent, although we have no right to deny their existence, since they have been frequently established (Czerny-Keller). Obviously, it is an essential characteristic of this condition, that it should involve a child born at full term and thoroughly healthy, who is nourished at the breast of mother or wet-nurse in accordance with all the fundamental principles which we have designated as rational and who is neither overfed nor insufficiently nourished. Under such conditions one sees at times that no regular thriving growth appears, in spite of the most careful observance of all the rules of nutrition, the administration of food being properly controlled by weighing, and the nurse being in perfect health. Instead, the children show restlessness, and dyspeptic bowel movements, of which the number is usually increased. The stools are of a varying appearance, usually green and slimy, more rarely pale yellow and harder, with a glistening oily appearance. The weight shows numerous fluctuations, with a pronounced tendency toward loss. Then, after we have reached a certain conviction that the feedings are neither too frequent nor too abundant, we proceed with the investigation of the nurse's milk, which usually reveals nothing abnormal either in the shape and development of the breasts and nipples, or in the macroscopic appearance of the secretion. This investigation does not always lead to a positive result, although we have at our disposal a number of reports, in which such a result was obtained, and treatment adapted to correspond with this result was effective. This failure to reach a definite result depends partly upon the fact that the technique of human milk examination is still largely incomplete, especially in respect to any proceeding which can be carried out in practice without great loss of time. Moreover the amounts of the various nutritive elements present numerous fluctuations of a rather wide extent, so that the analyzing of particular parts of a feeding is of no reliable value.

The negative results of the microscopic and chemical examinations of milk in certain cases preclude our finding the cause of the disturbances in the child, and only show, as Epstein pointedly remarks, that our present methods are not sufficient for a certain diagnosis of the abnormalities present. We must therefore with Epstein, Heubner,

and others, assume an idiosyncrasy of the infant toward this milk, or perhaps, as I have repeatedly seen, it may be an idiosyncrasy of the mother. Such an assumption can be proved with logical certainty, because a change of nurse, all other conditions remaining entirely unchanged, rapidly banishes the disturbances of digestion.

Nordheim discovered that Storch's reaction was absent in a case of this kind; its significance was rightly disputed by Thiemich. Beside these observations, which are always of enigmatical significance, there are cases in which the microscopic and chemical examination of the milk gives positive information. Thus, the presence of numerous so-called fine-granular milk globules is evidence of the bad quality of the food. The condition which is relatively most frequent is the finding of an abnormally large percentage of fat in the milk, which surpasses all normal fluctuations, and causes a corresponding increase in the movements of the amount of fat which can be recognized macroscopically. Thus, Budin and Michel found 50–115 grams of fat to the litre instead of 35, and the fat content of the stools was 35–65 per cent. instead of 20 per cent. Jemma found 65 grams to the litre. Quintrie and Guiraud have collected nine similar observations. De Rothschild describes several cases of this kind and cites a child observed by Variot and Méry, who showed such severe gastric symptoms that the writers thought of congenital pyloric stenosis, until a simple change of wet-nurse almost instantaneously stopped the vomiting. The other constituents of milk appear to play a less important part in this connection, although such cases have been reported (Marfan, Leviseur and others).

The diagnosis of this condition is always difficult, and is based upon the exclusion of injuries to nutrition in the method of nursing, the absence of an infectious factor, and the possible positive results of the examination of the milk. As a procedure for the rapid diagnosis of a case of this kind, Marfan recommends that the child be taken from the breast for 24 hours and nourished with sterilized cow's milk. Improvement or cessation of the symptoms during this period points to the breast-milk as the cause. This procedure does not always necessarily attain its end, because many infants, especially young babies, react severely to cow's milk. Consequently I would rather recommend the use of the breast of another woman if it can be done.

When the examination shows no qualitative changes in the milk, and when the symptoms do not improve after prolonged observation, a wet-nurse or change of wet-nurse is indicated in the early months of life, and gradual weaning in the later months of life, the latter all the more as the interpolating of cow's milk feedings often has a favorable influence upon the condition.

When an abnormal richness in fat is believed to be the probable cause of the unsuitability of the milk, one can try putting the child

to both breasts each time and not allowing it to empty them, in order thus to shut out the last part of the milk, which is richest in fat. Or, feedings of whey can be interpolated between the breast-feedings, or, in accordance with the proposal of Quintrie-Guiraud, cow's milk can be given alternately with the breast, diluted one half with lime water, and with milk-sugar added in the amount of 35 grams (one ounce) to a ½ litre (pint) mixture.

The other abnormalities, such as increased percentage of casein or of salts, are so infrequent that no general principles of treatment have been established. In individual cases a choice must be made between change of nurse, weaning, or mixed feeding.

D. **From Insufficiency of the Digestive Organs** (may result from premature birth, hereditary taint, intra-uterine infection, malformations of the digestive apparatus, mechanical obstacles to sucking).

This is not the place to give a complete description of *premature infants*, consequently I shall limit myself to discussing those factors which constitute the cause of the abnormal course of their digestion, and the occurrences of diseases of nutrition. There can be no serious doubt, even if the evidence is only of an anatomic character, that children who come into the world months before the normal termination of pregnancy, present actual insufficiency in regard to the absorptive and assimilative power of their digestive organs. The defective differentiation of the secretory elements of the gastric glands, the shortness and width of the intestinal crypts, the embryonic character of the liver structure, the small size of the salivary glands, and so forth, point with certainty to this conclusion. To preserve such infants requires proportionally more abundant nourishment, as does also their protection against loss of heat by radiation, which is especially active on account of the relatively large area of their skin surface, and in consequence of which more food is required to furnish increased activity in the functioning of the underlying chemical sources of heat. The fact that the active taking of food by sucking is often impossible, so that feeding by means of milk squirted directly into the mouth or poured in with a spoon, vessel or tube, or even perhaps through the nose, must be resorted to, is a further reason for digestive disturbance, in the form of a reaction on the part of the extremely sensitive organism. Finally, the small capacity of the stomach necessitates an increase in the number of feedings and a corresponding shortening of the intervals in order to give sufficient food. This constitutes a factor which easily leads to overfeeding, the results of which are more easily brought about, and are much more noticeable in their intensity, in premature infants than in the fully developed. There is a general agreement as to the necessity of supplying an increased number of calories. Thus Budin states that premature infants must take up to one fourth of their body

weight of mother's milk a day in the first ten days, and after that require about 20 per cent. of the body weight. Even such strict observers of dietetic principles as Czerny and Keller recommend 110–120 calories per kilo, while Finkelstein recommends 140, and they all with one accord point out the danger of an insufficient amount of food. Czerny and Keller seek to avoid overfeeding by prolonging the intervals and when the amount of milk taken by the child of its own accord is insufficient, they instill the rest artificially, while Budin, Marfan, Finkelstein and others designate 9–12 or even 24 feedings as necessary.

If we consider the further fact that the cooling off of the milk, which is hardly to be avoided in artificial feeding, can itself form a source of irritative symptoms, it can easily be understood in view of all the above-mentioned facts, that premature infants often react to these efforts at preserving their lives with severe gastro-enteric symptoms. These soon manifest themselves in the form of vomiting after every feeding, which is of special danger on account of the easy possibility of fluid gaining entrance to the air-passages, and in the form of diarrhœa, which sometimes shows numerous green slimy movements, and sometimes light yellow fatty stools. These symptoms lead to disturbance of the mechanism regulating temperature, against which the most careful regulations of the temperature of the infant's surroundings proves powerless. It leads also to relatively tremendous falls in weight, and to the development of secondary infections, for in such children the portals of infection even normally stand half open, and the power of self protection is not enough to guard against it.

For all these reasons the *feeding of premature infants* requires special care. They should whenever possible, be fed only on breast-milk, and the active taking of the food by sucking should be furthered in every possible way, such as by stimulating rubbing, or by giving a mixture of tincture of valerian, ether, and distilled water in equal parts, dose 2–3 drops immediately before feeding. We endeavor as far as possible to reduce the amount of heat required from the food, so that we can get on with smaller additional quantities artificially administered. This is effected by a sufficient and continual supply of external heat, which is best attained in an incubator, out of which the child is never taken even for feeding. Moreover, for the first 10–12 weeks at least we supply a nurse whose freely flowing breasts and easily grasped nipples are adapted to the requirements of a weak infant. Also we can try, as recommended by Budin, the administration of pepsin, of which a little piece of a tablet is crushed and put in a teaspoonful of alkaline water such as Carlsbad or Mühlbrunnen, and given before nursing.

If in spite of every precaution disturbances of digestion have appeared, an especially careful treatment is required. This consists in the first place of a constant supply of heat, and a carefully regu-

lated diet. As to the latter, I should like to recommend the vegetable broth recommended by Méry on account of its effective results against loss of weight. Its composition and method of use will be spoken of later. The treatment also consists in stimulating baths, with the addition of powdered mustard, rubbing with alcohol, and subcutaneous injections under strict antiseptic precautions of small quantities of physiologic salt solution or Hayem's serum, also described later in detail.

No use is made of drugs, except as stimulants. In addition to the valerian drops mentioned, caffeine citrate or sodium benzoate may be given in doses of .01 Gm. ($\frac{1}{6}$ gr.) three or four times a day.

A further cause of insufficiency of the digestive organs is hereditary weakness from tuberculous, syphilitic, or alcoholic parents.

Their descendants do not necessarily inherit the signs of the disease, but its influence frequently manifests itself in the creation of an offspring, which, although born at full term, is very backward in physical development, and is afflicted with such a deficient functioning power of the gastro-intestinal canal, that the slightest deviation from the strict principles of nutrition leads to severe alteration in its function. These find their expression rarely in acute, more often in chronic disturbances of digestion, which terminate in cachexia. It is important to recognize the inferiority of such children, in order to supervise their nutrition from the start with the greatest rigor, and to offer them at least some prospect of preservation of life. That manifest hereditary syphilis leads to weakness of digestion is well known, and it will be considered in detail in another part of this work. Intra-uterine infectious processes occur, although they are in fact great rarities. If they do not terminate the life of the fœtus, they result in peritoneal adhesions. They can affect the digestive power of the child, partly by obstructing development, and partly in mechanical ways by the formation of stenoses, kinks, and similar affections of the intestine. They constitute a source of occasionally acute but mainly chronic disturbances, the treatment of which consists in careful hygiene and well managed breast-feeding. Surgical measures may have to be added as an auxiliary. We will make further consideration of these forms when treating of congenital intestinal stenosis and atresia. Also certain malformations of the digestive apparatus, particularly dilation of the colon (Hirschsprung's disease, or megacolon congenitum) will be treated thoroughly later. Here we will speak only of those affections which influence nutrition by mechanical interference with sucking, and which, perhaps, can have an injurious action through destroying certain protective mechanisms. Harelip is usually no reason for the inability of the child to take the breast, and consequently it does not compromise nutrition. Also cleft palate, involving simply the soft palate is compatible with a shutting off of the nasal cavity from the buccal,

and permits the act of sucking. On the other hand complete cleft palate forms an absolute obstacle to the active taking of food from the breast, and such children must be fed with a spoon, a tube, or in some similar way. In such cases, catarrhal processes of the pharynx almost always develop, and these are favored by the absence of filtration of the air in passing over the nasal mucous membrane. These catarrhs soon extend by contiguity to the stomach and intestine. It is therefore no wonder that such patients show severe disturbances of nutrition, and that in addition to the insufficiency of milk taken, vomiting and diarrhœa supervene, resulting frequently in death in a relatively short time.

In a similar way local processes in the mouth of an inflammatory nature can be injurious, since on the one hand they render the act of swallowing difficult and painful, and on the other hand they give rise to an extension of the process to the mucous membrane of the lower alimentary canal. I mention particularly in this connection catarrhal and aphthous stomatitis, both of which processes require especial attention in early life and must be carefully treated, to avoid these results.

Tongue-tie is frequently blamed as an obstacle to sucking, and the cutting of the shortened frenum is still performed by many physicians. It has no influence on sucking, inasmuch as we know from investigations on the physiology of the sucking movements, that the muscles of the floor of the mouth, the lips, the cheeks, and the soft palate play the dominant part, while the tongue remains quiet and serves as a conduit for the passage of the milk, a function which cannot be interfered with by a short frenum. The desire that the frenum be cut should be energetically combated, because such a measure can be followed by bad results.

On the other hand, sublingual tumors of notable size and rapid growth can form a real obstacle to the acts of sucking and swallowing, and therefore such conditions must be relieved by operation.

Also facial paralyses, which not infrequently develop as a result of difficult labor, especially when forceps have been used, are an obstruction to sucking worthy of attention and compel us to carry out the nutrition of such children in the early weeks by artificial measures. Fortunately this paralysis is usually transitory, and by energetic treatment with electricity we can soon bring about the appearance of normal sucking power.

Narrowing of the nasal cavity is caused or favored by a peculiar anatomic arrangement of the arch of the palate, which is of high and narrow structure. With the occurrence of catarrhal swelling of the mucous membrane such a narrowing can lead to complete obstruction of the passage, and often constitute an obstacle to nutrition which is severe and highly deserving of attention. The child during nursing is unable to breathe on account of the nasal obstruction, and constantly

lets go of the nipple. Finally, tired by the exhausting effort, he refuses the breast altogether, and consequently takes an entirely insufficient amount of food. Such a condition requires local treatment, such as cleansing with a lukewarm boric acid solution, cauterization of the mucous membrane with a 4 per cent. or 5 per cent. silver nitrate solution, or perhaps even the introduction of drainage tubes while nursing. In syphilitic infants applications are made of tampons with blue ointment or yellow precipitate ointment. Besides these measures, the breast can be administered indirectly by means of a sucking apparatus, such as those proposed by Bouchut, Aurard, and others. They are of such construction that the nurse by sucking through a tube aspirates the milk from the breast into a glass vessel pressed about the nipple, while the child drinks from the vessel through a second tube. These implements are hard to keep clean, and in spite of all precautions such as cotton plugs the aspirated milk is very easily contaminated by the saliva of the nurse. This leads to disturbances of digestion in the child which soon counterbalance the advantages of more abundant food. It therefore appears preferable in such cases to express the nurse's milk and give it from the bottle or with the spoon. The natural method of sucking should be restored as quickly as possible by energetic treatment of the nose.

E. **From Bacterial Contamination of the Food.**—These forms, which as we shall see have been long recognized, and play an exceedingly important part in artificially fed infants, are found relatively infrequently in exclusively breast-fed infants. Positive diagnosis is based upon the fact that the child gets nothing but human milk to drink and upon the demonstration of the exciters of the infection in the food, and in the stools. Human milk is not easily subjected to bacterial contamination, on account of its method of production by the mother and of abstraction by the child. It is very possible that during the progress of certain general infections, such as puerperal processes, severe pneumonia, and similar affections, their exciting agents are excreted through the mammary glands, although usually such diseases so affect the general condition of the mother that she quickly loses the power of nursing her infant. On the other hand, local processes in the breast, especially infections of the glandular substance resulting from fissures of the nipple and leading to abscess-formation can contaminate the milk, and it is a question whether infectious diseases of nutrition cannot arise in this way.

Damourette, who has done thorough work on this subject, is inclined to assign to this fact of milk contamination an importance which in my opinion is rather excessive. He cites in his monograph a large number of instances, in which digestive disturbances varying from a mild to the most severe and even fatal character appeared after the

taking of such "pus-milk." But a closer analysis of his cases shows that they did not conform to the postulates mentioned at the outset as requisite, in that neither the condition of exclusive breast-feeding, nor of avoidance of overfeeding, nor finally of the demonstration of the exciting organisms in the breast-milk and movements, were fulfilled. Epstein frequently had the opportunity of observing infants who for days and weeks were swallowing pus from suppurative processes in their mouths, particularly abscesses of the salivary glands. He was never able to establish the fact of any influence from this cause upon nutrition, although such cases are not significant in this class of diseases because the infectious material came from the child's own body, and therefore there can be a certain auto-immunization against it. On the other hand, Moro has described a streptococcus enteritis observed by him in a number of infants exclusively nourished at the breast of a thoroughly healthy nurse, in a rational way. These cases fulfil all requirements and are a proof that diseases of nutrition can occur in this way. Through a number of researches which have been collected in the above-mentioned brochure of Damourette, we have learned that the fact of the sterility of normal human milk is only true with certain limitations, since the portion that first flows out of the mammary gland shows a variable bacterial content. These organisms are mainly the staphylococcus aureus and albus, less often the streptococcus, the bacillus coli communis and others. They probably gain entrance from the surrounding skin by way of the collecting ducts of the nipple. In view of the fact that they have no pathogenicity toward animals, and produce no lesions in the mammary tissue, no significance has been attributed to them, and it has been assumed that the protecting power of the digestive tissue of the infant is sufficient to render them harmless. Moro, pursuing these investigations still further has shown that if cultures are made from sufficient quantities of milk, pus organisms, particularly the staphylococcus pyogenes albus, can always be demonstrated in the portions of the milk pressed out of the emptied breast after careful cleansing of the nipple. Less often he found the aureus and the bacillus acidophilus; occasionally also mycelium and sarcinæ.

He observed in a number of children the clinical symptoms of an acute intestinal catarrh coming on suddenly during full health which led to the passage of spurting movements of a light yellow color with a slight greenish tinge, and less often to the production of a flocculent slimy diarrhœa of small masses of mucus expelled with tenesmus. These cases always ran a favorable course. Microscopic examination showed in preponderance fat globules, fat crystals, fatty soaps, glairy mucus, intestinal epithelium and in two cases there were also numerous leucocytes. Cover-glass preparations stained by Weigert's method showed,

instead of the normal bacteriologic picture, only occasional bluish black bacilli, and in every field numerous dark blue-stained clumps of staphylococci and a few somewhat larger cocci, also Gram-staining and arranged in pairs of short chains. Cultures from the stools gave the characteristic colonies of the aureus and albus, which showed no pathogenicity toward animals. The same species were to be found more or less abundantly in the milk of the nurse. An experiment of Moro's on a child seems to me especially significant. When a child of previously normal digestion was put to the breast of a nurse in whose milk numerous staphylococci were found, it promptly reacted with dyspeptic movements showing the characteristic bacteriologic finding.

Rosthorn briefly reports an epidemic of intestinal catarrh, also in purely breast-fed infants, observed in the Heidelberg lying-in hospital, in which epidemic a staphylococcus was found in the stools. These observations were not so free from objection as those of Moro, because in the first place the patient showed various skin affections with the consequent possibility of infection from another source, and in the second place the microörganisms were not demonstrated in the milk.

Nevertheless, such cases suffice to point out the possibility of the occurrence of diseases of nutrition after taking breast-milk contaminated from without by suppurating organisms derived from the nipple, and point the way toward prophylaxis. We should retain the custom of rejecting the first portions of milk and of carefully cleansing the nipple before putting the child to the breast, a custom which was formerly general, but which has lately been largely considered superfluous. The same consideration will cause us to forbid further nursing from an inflamed breast, which is also prohibited for other reasons pertaining rather to the nurse.

The treatment of such infections is relatively simple. When there are severe enteric symptoms, we put the child on a water or weak tea diet for 12–24 hours, and in the meantime treat the breast by repeated mechanical emptying and by careful cleansing of the skin with corrosive sublimate, alcohol and ether. If this does not bring about a marked diminution or a disappearance of the bacterial content of the milk, we seek a better qualified nurse. For the diarrhœa, Moro recommends rice jelly, to every 50 grams of which a gram of tincture of opium is added, and gives a teaspoonful of this mixture every two hours.

SECONDARY GASTRO-ENTERIC DISTURBANCES. SEPTIC INFECTION WITH GASTRO-INTESTINAL SYMPTOMS.

It still remains for me to discuss very briefly the gastro-intestinal disturbances developing in connection with other diseases of infancy, and appearing as complicating symptoms often of a severe character, and also the gastro-enteric symptoms forming the clinical manifesta-

tion of a general septic infection. The infantile intestine, on account of its marked irritability, reacts easily to diseases located in other organs, especially if such diseases are of an infectious nature and run a febrile course. For example it is a known fact that influenza in early life is often marked by severe gastro-intestinal symptoms, the proper interpretation of which encounters difficulties. Frequently, only the additional presence of rhinitis, the spreading of influenzal diseases in the family or among the inmates of the sick-room, and the prevalence of an epidemic puts us on the right track. The same is true of febrile catarrhs of the respiratory tract, such as rhinitis, angina, and bronchitis, which cause involvement of the gastro-intestinal canal either through the action of absorbed toxines, or through direct extension of the process, or through the swallowing of the secretions. Such cases are characterized by the appearance of frequent movements containing mucus, and often of mucous vomiting, the latter symptom especially being an indication for stomach washing. In the same way the different acute infectious diseases of early life, such as diphtheria, measles, scarlet fever, German measles and whooping-cough, are often accompanied by vomiting and diarrhœa, which have an unfavorable influence on their course. Also when the reaction does not appear as the direct result of the infection of the body, secondary disease of the gastro-enteric tract may come on as a result of the anorexia and weakness of digestive power accompanying these diseases, in combination with too abundant feeding. This retards convalescence and if it is very prominent, may eventually assume threatening proportions. The feeding of children with a febrile infectious disease should be carefully adjusted to the weakened function of their intestine, with long intervals between the feedings and reduction in the amount given each time. What trivial influences may cause severe disturbance of digestion is shown by the appearance of such symptoms after relatively slight surgical operations, or after strapping children down in the beds devised for the collection of urine and feces in metabolism experiments, or after shutting them up in the respiration apparatus (Epstein, Bendix, Rubner-Heubner). This leads us naturally to those cases which are apparently gastro-enteric affections, but which are really the clinical manifestations of a septic infection finding its entrance to the body in an entirely different place. This is not the place to consider these interesting forms of diseases and their diagnostic importance. I refer the reader to my article on this condition in the "Traité des maladies de l'Enfance," in which are explained the reasons for my introducing the term "Sepsis with gastro-intestinal symptoms" for this condition, a term which in my opinion characterizes the nature of the process with sufficient definiteness. I will only mention here that even with the most accurate clinical observation it is often impossible to reach the right diagnosis with cer-

tainty, and this is only attained by autopsy and thorough histologic examination. I do not deny the possibility of a general septic infection originating in the intestinal canal itself, but I declare this to be an occurrence exceedingly rare in breast-fed children, and to be hitherto unsupported by reliable observation.

VII. DISTURBANCES OF NUTRITION IN ARTIFICIALLY FED INFANTS

A. **From Overfeeding.**—It is easy to understand how this condition is especially liable to occur with artificial feeding. In the first place, our knowledge of the physiology of infant feeding apart from the maternal breast is so incomplete, that no normal standards have been established, and it is usually from lack of success that we first conclude that we have not chosen the right way (Czerny-Keller). Then the mode of administering the nutrient fluids or mixtures is of such a nature that it calls for no labor on the part of the child, whereas nursing from the breast ceases after a certain time through the coming on of fatigue. Furthermore in breast-feeding there is always an adjustment of the composition of the food to the temporary requirements of the child, as a result of which a lessened concentration of the breast-milk corresponds with an increased amount taken. This adjustment is lacking in artificial feeding, for in food prepared artificially the concentration either remains the same, in the more favorable cases, or else, more commonly, is increased. There are a number of additional factors. There is a tendency on the part of those in charge of the child, to give it as it grows older relatively rapidly increasing quantities of food without regard to its appetite and actual needs. The intervals, which should be very long to correspond with the slower course of the process of digestion, are made much too short. The restlessness so often present on account of the shorter and lighter sleep of artificially fed infants is interpreted as hunger, and is quieted by the constant giving of additional food. There is a prevailing custom, especially common among the masses, of letting the child lie in its bed by the hour with the nipple of a half-filled bottle in its mouth. All these facts, together with the uncertainty prevalent among physicians as to the proper amount and dilution of the food used, form a sum total of reasons which make it very easy to understand why in artificial feeding permissible limits are so easily exceeded. Further, the power of assimilating animal milk appears to vary with individuals (Czerny-Keller), so that what is rational with one infant may be excessive with another.

Finally there is the alteration of the intestinal flora first mentioned by Tissier, which conduces to abnormalities of the process of digestion, because new types appear which force into the background those normal varieties which are auxiliaries to the physiology of digestion. The complete neutralization of the free hydrochloric acid by the

increased content of salt in cow's milk, together with the too frequent and too abundant administration of this food, exposes the gastric content to all possible bacterial contaminations and causes the passage into the intestine of a greatly altered material, of which the irritative action soon becomes noticeable. The work thrown on digestion, varying with the individual but always excessive, is still further increased by excess of food, leading to stagnation of the milk in the stomach, development of abnormal fermentation, and production of abnormal gases.

The **symptoms** of these injuries become first noticeable through vomiting, which soon appears in young infants nourished by cow's milk even with slight overfeeding, and which is characterized by great obstinacy. The difference in the coagulation of the proteids in human and animal milk is shown in the appearance of the vomitus, which contains large curds floating in grayish white, less often in bile-stained, serum. On account of the more marked diminution of the gastric motility the expulsion of these curds is effected by the child with greater difficulty, which finds its expression in the appearance of pallor before the occurrence of vomiting, in restlessness, and frequently in the projecting to some distance of the flood of fluid. Then, rather rapidly, —much more so than in overfeeding in breast-fed infants,—there occur a poor general appearance, the suffering expression of the face, the discomfort, the loss of sleep, and all these are much more pronounced, and of a much more severe character. The formation of gas in the stomach leads to belchings of a bad odor, the tongue quickly becomes more or less extensively coated, the abdomen prominent, the nature of the child changes, its cheerfulness disappears, its interest in its surroundings diminishes, the movements of its limbs become less, in short one gets the impression that the disturbed digestion has greatly injured it physically. Its tissues, especially its muscles, feel softer, and its urine passed abundantly, decomposes rapidly and imparts to the napkins an extremely disagreeable ammoniacal odor. It is always free from albumin, but is rich in indican. The intestinal discharges may at first show but slight deviations from the normal, so long as the stomach is the chief seat of disturbance. They show a pulpy consistency, an ochre-yellow color and but slight fecal odor, and exercise no irritative action upon the skin of the anal region. Usually, however, it is not long before the altered chyme begins to cause disturbance in the intestine. It irritates the mucous membrane through the products of its fermentation, excites increased secretion, and thus peristalsis is quickened, and the undigested residue of the food is hurried along and out in more abundant quantities. The number of movements are increased to 4–6 in 24 hours, their consistency becomes semi-fluid, they leave the anus with a noise, make broad wet spots on the napkin, present an abundant admixture of mucus, and of larger and smaller white fragments as

well as greenish spots and streaks, and they give off an odor at times disagreeably pungent, at other times notably foul.

In the meanwhile the subjective discomfort increases, the distention of the abdomen becomes greater, and an important sign of more marked development of the injury is seen, in that the appetite diminishes so that the bottle is only occasionally emptied, or is rejected after a few sucks.

The *weight* usually continues to increase at first, and at times may even reach an abnormally high point, in which case the weight curve shows a more irregular course and is now and then interrupted by rather sharp falls. But as soon as the diarrhœa appears, or just before, the weight begins to be stationary, and soon afterward a fall begins which is slight at first, but which may suddenly change to a rapid loss of weight. The suddenness with which these disturbances take those around the child by surprise is one of the peculiarities of the diseases of nutrition in the artificially fed, and consequently constitutes their great danger, because attention is attracted and medical aid invoked at a time when such aid comes too late, or when it can only ward off a bad termination with great difficulty. The earlier in life such processes occur, the greater and more far-reaching are their injurious results, and the longer time do they require for their eventual cure.

We will pause at the first steps in their progress, and speak briefly of the **treatment** indicated in this stage. The indications may be briefly summed up, first, the removal of fermenting and decomposing food from the stomach and intestines; and second, the institution of a method of feeding which shall require as little as possible from the weakened power of digestion, especially as the danger of giving too little food is much less than that of giving too much. The first requisite is attained by a bland diet consisting of water or weak tea, which is preceded by a thorough washing out of the stomach since in contrast with the analogous condition in breast-fed infants, simple withdrawal of food is not enough to relieve the vomiting. The emptying of the stomach, even many hours after the last feeding, produces large curds which easily clog the opening of the tube, and considerable tenacious stringy mucus. If the tea or water is still vomited, the washing must be repeated on the following days until the fluid given is tolerated. If loose movements of bad appearance and bad odor are present, irrigation of the intestine is useful. For this purpose a graduated glass irrigator is employed, having a rubber tube about three feet long, which is connected by means of a stop-cock with a Nélaton catheter. The child is placed on its side with its legs drawn up against the body, and the thoroughly oiled tube is introduced to three quarters of its length. Then the irrigating fluid (boiled water, 2–2.5 per cent. boric acid solution, or normal salt solution) to the amount of $\frac{1}{2}$–1 litre ($\frac{1}{2}$–1 quart), according to the age of the child, is allowed to flow in.

Medicinal treatment is, to say the least, superfluous. The water diet can be continued several days without hesitation, especially as the children soon become quiet and obtain undisturbed sleep. The complete cessation of the vomiting and the appearance of infrequent small movements almost wholly without odor signify that the time has come for attempting cautiously a resumption of the feeding. A further sign is found in the condition of the tongue, which, after the removal of the decomposed material from the alimentary canal, quickly begins to become clean, from the tip backward. While it is easy to resume feeding after a relatively short period in overfed nursed infants, it must be done carefully when artificial food is given. In very young infants, which show the typical results of feeding with cow's milk, it is advisable to make no further trial of this food, but to put them to the breast of a wet-nurse. When, for various reasons, this is impossible, one should begin with very small quantities of much diluted milk prescribing long intervals between the feedings. At intervals of at least four hours one adds a teaspoonful of milk to the fluid given at each feeding, and gradually increases this amount until convinced from the absence of vomiting and the good condition of the movements that the food is well borne. Unfortunately, as we have already mentioned, we have no normal standards at our disposal as we have in breast-feeding, by which we can regulate the further feeding of such children, and therefore have to feel our way experimentally in each case, a process which requires much patience and continued observation. We must be guided by the function of the stomach and intestine, the appearance of the child, its behavior, its sleep, and most important of all by the weight curve, which should no longer fall when a little food is given, but should begin slowly to rise when the smallest amount of food above the minimum required for the maintenance of life is given. With each disturbance we go back, either in the amount or concentration of the food, or in both. We can try in these cases to support the stomach by medicines aiding digestion (as pepsin in powder or tablets, pancreatin, or hydrochloric acid, $\frac{1}{2}$–1 per cent. solution given in teaspoonful doses), or by choosing preparations of milk in which the child is spared part of the work through partial predigestion of the casein. Also small doses of alkaline water work well, particularly Carlsbad and Mühlbrunnen, given slightly warmed in doses of 1 to 2 tablespoonfuls each morning.

Overfeeding with cow's milk is not necessarily limited to the results described above, but may often after a relatively short time and without any particularly clear warning signals, lead to disturbances which are much more severe and proportionately much more difficult to alleviate. The symptoms of vomiting and diarrhœa come on with increased severity, in that on the one hand the vomiting undergoes a postponement in time and appears relatively long after feeding, and

on the other hand the movements become larger and more frequent, and show more mucus, a broader spot on the napkin, a looser consistency, and a few gray or greenish yellow fragments. The secretion of urine diminishes, the temperature rises, the general condition becomes worse, in that constant restlessness, permanent crying and a half waking condition of the child hardly to be called sleep develop, and the loss of weight is rapid and constant. The gravity of the situation is obvious even to the eyes of the laity, from the visible emaciation of the body, the expression of anxiety and suffering, the dry and frequently protruding tongue, and the feeling of thirst with all its manifestations. On closer examination we find a slight depression of the fontanelle, the skin is pale and feels hot and dry, the thermometer shows a temperature of 39°–40° C. (102°–104° F.), the mucous membrane of the mouth appears red and swollen, and in young children spotted with thrush, the tongue shows enlarged papillæ, and a more or less wide-spread dry coating of gray or dark brown, the eyes are expressionless, and their glance weak, the neck is visibly thinner, the respiration is accelerated and often of an irregular type, the abdomen is distended and the peristaltic movements show through the skin of the abdominal wall, the thighs feel pulpy and their covering of skin appears too large for them. The skin of the genital region shows redness, swelling, ulceration, or nodule formation. Râles are to be heard in the lungs, especially at the bases. The appetite appears notably lessened, for in spite of the tormenting thirst, the bottle is rejected after a few swallows, and often nursing is followed by severe colicky pains which precede the passage of a small light-colored movement. The rapid and frequently unexpected appearance of these symptoms, the fever, the marked affection of the general condition, the irritative character of the movements, the loss of weight, and so forth, all signify that in addition to the overloading of the digestive organs another factor, that of endogenous infection and intoxication has appeared which requires speedy relief. The indications are for immediate suspension of feeding, quenching of thirst with water or tea, which with high fever and a marked tendency to vomit should be given cool, and for a moist pack of the trunk or of the entire child. For the latter a cloth is folded to a thickness of four layers, is wet in water at the room temperature or up to 20° R. (77° F.), is thoroughly wrung out, and wrapped around the child, and over it is put a flannel cloth. This procedure can be repeated every quarter to half hour, but should not be used oftener than three or four times in succession. If there is great restlessness, a lukewarm bath may be tried. When the vomiting is not marked, or if it ceases after the starvation-treatment, we can hasten the evacuation of the bowel by means of calomel, which I give preferably in small, frequently repeated doses. This is however unreliable, and the most efficient treatment is by diet, in connection with

intestinal irrigation. If the temperature is very high, it can be effected with cool water, and in such cases the use of 0.7 per cent. salt solution is advisable, as it is partly absorbed by the mucous membrane, and so helps the washing out of the entire organism; besides that, it soothes the intestinal epithelium injured by the inflammatory process. Under this treatment, the fever usually falls, and a quieting effect is produced, which is evidenced by more prolonged sleep, and by the return of a peaceful expression, which delights those around the child. The vomiting, which finally consisted simply in retching producing only a few drops of fluid or a little mucus, now ceases. The general appearance improves, the movements become less frequent and take on the typical characteristics seen on a water or tea diet, the secretion of urine increases very markedly, an especially favorable sign; the mental alertness returns, the glance becomes freer, the play of the features more active, in short, reaction has appeared, and the danger is removed for the moment.

An unfavorable change in these cases occurs, in the first place, when from increase of the gastro-intestinal symptoms such weakening of the body comes on, that it succumbs to secondary infections, which are usually situated in the region of the lungs. The fever, already fallen, quickly rises again to its original height or exceeds it, the breathing becomes more rapid and visibly labored, with movement of the alæ nasi and muscles of the neck, the chest is expanded with difficulty and in its lower part is drawn in with inspirations. The pallor changes to an obvious cyanosis or to a grayish blue tint which is especially ominous, the pulse becomes impossible to count and difficult to feel, and the heart action weakens, as is shown by the muffling of the heart sounds. One can usually find areas of consolidation on one or both sides at the base of the lungs near the spine or more widely distributed. This scarcely causes any cough, because the muscles of respiration are so much affected that they are evidently not strong enough. Consciousness becomes clouded, the mouth is held open and shows a dark red, dry mucous membrane. The eyes show infrequent movements of the lids, the dull corneæ show superficial ulceration, and the conjunctivæ show hyperæmia and mucopurulent secretion. A weak monotonous whimpering replaces the former painful crying, and reveals the gravity of the situation to anyone who has once heard it. The stools trickle out of the open anus, and now and then a brief convulsive twitching suddenly traverses the body, or a deep sigh interrupts the stupor. In such a condition life ceases. The prognosis in such a course of events is very unfavorable. Stimulant procedures are indicated. Mustard baths may be used, prepared as follows; a linen bag is filled with one fourth of a kilo (half a pound) of powdered mustard, and is swished back and forth for several minutes in water at a temperature

of 28–29° R. (95–97° F.). One may try warm packs with friction of the body, or injections of caffeine [.02 gram ($\frac{1}{3}$ grain) of caffeine citrate or sodium benzoate]. Ether may be injected under the skin near the heart, although this usually results in necrosis, or one fourth of a syringeful of camphorated oil may be injected. All these measures can only temporarily assist the weakened condition of the child, as their action quickly ceases and the former condition returns. Strong expectorants, such as liquor ammonii in 1 per cent. solution in teaspoonful doses every hour, are usually but little effective, especially as the power of swallowing is affected, so that the medicine either flows out again or causes vomiting, and there is danger of entrance of the fluid into the respiratory tract.

Another course of events is seen when there is a constant increase in the severity of the gastro-enteric symptoms. The vomiting becomes constantly more frequent and is replaced by continual retching, and the movements lose their fecal character and eventually consist only in gray or reddish fluid in which float little grayish white specks, which microscopically are seen to be intestinal epithelium. The temperature constantly falls till it reaches a subnormal degree, while the temperature of the interior of the body always appears higher than that of the surface, and finally the typical picture of *cholera infantum* develops. The large fontanelle appears depressed, the eyes, surrounded by dark circles, are deeply placed in their sockets, the cold pointed nose projects prominently from the face, the expression presents a mixture of suffering and moribund, the body seems ice-cold, the skin is covered with a clammy sweat, the abdomen is sunken, and the limbs are stiff and pressed against the body. The skin of the lower leg, and frequently of the thighs and buttocks feels hard, as if its subcutaneous tissue were filled with a stiff mass (Heubner), and folds of skin when pinched up remain prominent. (This condition has been designated by the name sclerema, and is connected with the hardening of the subcutaneous fatty tissue, as a result of the marked fall of temperature.) The passage of urine stops entirely, or else a few drops of cloudy urine are expelled with difficulty, which contain much albumin and many formed elements, especially casts of various kinds, leucocytes, and renal epithelium. The stools are either passed involuntarily, or flow almost continuously from the anus. Consciousness has almost disappeared, the child lies weak and exhausted with the head deeply sunken in the pillow, its mouth exhales a stale odor, its faint cool breath is gruesomely suggestive of the approaching end. From time to time, this comatose condition is disturbed by brief twitchings or more prolonged general convulsions, until finally such an attack ends the scene or else the heart action gradually ceases and death occurs imperceptibly. The fully developed symptom-complex of cholera infantum is almost

invariably fatal. Only in rare cases is it possible, by means of hot baths [35° C. (95° F.) or more, with or without mustard] followed by dry warm packs, and hot water bottles, to rekindle the fading spark of life. All mechanical procedures, such as irrigation of the stomach or intestine are in this stage not only useless, but directly dangerous. On the other hand, if fluid cannot be administered by mouth or rectum, one can try *subcutaneous injection of salt solution,* or of the so-called artificial serum [3 grams (45 grains) of sodium bicarbonate, and 4 grams (one dram) of salt in 1 litre (one quart) of water]. This procedure is performed under strict antiseptic precautions. Either a funnel is used connected by a tube with a needle which is stuck deep into the skin of the lower abdominal region, or a large syringe is successively refilled and adjusted to the cannula, which remains sticking in the skin. The former method is the more convenient, and therefore preferable. The fluid is injected at body temperature, in an amount varying from 50 to 100 c.c. ($1\frac{1}{2}$ to 3 ounces) according to the severity of the case and the intensity of the loss of waters; the procedure is in the case of necessity performed twice a day. When the marked weakening of the heart results in a notable retarding of the absorption of the solution, one may attempt to hasten it by massage. Usually, however, absorption takes place in a relatively short time, without injury to the skin, although it is distended and rendered anæmic by the fluid. These injections increase the strength of the heart, and check—at least, partially—the loss of the fluids which dilute the poisons circulating in the body and favor the excretion of these poisons by stimulating the renal secretion. The immediate effect of these injections is often very noticeable, in that the stupor disappears, the general appearance improves, the pulse becomes stronger, the passage of urine is resumed, and in short the impression of a very severe illness disappears for some time. In relatively rare cases, it is possible by constant repetition of this procedure to cause a lasting improvement of the condition, so that voluntary taking of fluid becomes again possible, and the fluid taken is retained. More often, however, the disease progresses, interrupted by short reactions becoming constantly less marked and the decline of strength cannot be checked. The scene does not always end in the way described above, for the comatose stage may be replaced by one in which the symptoms point to a marked affection of the brain and meninges. This manifests itself clinically in contractions and tonic rigidity of the extremities, which lie half flexed against the body, in very marked rigidity of the neck, in shrill outcries, and in irregular breathing, which shows either the Cheyne-Stokes type, or deep sighing respiration as in diabetic coma, or an alternation of this or that irregularity. The picture reminds one of the advanced stage of tuberculous meningitis (hydrocephaloid of the older authors).

Again, the symptoms referable to the stomach and intestines may be less prominent in comparison with the severe general intoxication, and the picture of collapse, algidity and dessication of tissue develops, without any mechanical explanation in copious vomiting or profuse diarrhœa (cholera sicca).

Finally the course may vary in that the storm abates a little, and instead of the violent symptoms, a febrile condition develops with severe prostration, in which the failing patients reject all food and drink, in spite of the fact that the brownish discolored lips, the thickly coated leathery tongue, and the deep red buccal mucous membrane covered with thrush, are evidence of great lack of fluid in the tissues. The vomiting has become less frequent, the movements have lost their choleriform character, are passed only two or three times a day, and consist of brownish yellow, slimy, not very copious masses with an extremely foul odor. There are usually râles in the lungs, and frequently also inflammation in the form of small areas localized along the spine or in the lower lobes. The urine is passed in small amount, and is dark yellow in color. The picture reminds one of the later stages of a severe case of typhoid fever (hence the name "cholera-typhoid").

There is no doubt that the participation of the kidneys rapidly increasing to anuria, may sometime dominate the disease picture; but this does not occur prominently enough to warrant the clinical differentiation of a uræmic type of the disease. Nevertheless, uræmic intoxication and insufficient elimination of the poisons from the body through the kidneys appear to play an important part in cases which come on with convulsions, and in the course of which convulsive movements are prominent, and in which at the same time the existing anuria or oliguria and the urinary findings indicate the severe involvement of the kidneys.

The treatment of cholera attacks we have briefly considered above, and we now have to take up the resumption of feeding and the further care of those children who have survived acute attacks. This treatment requires very especial care, and in my judgment can only be carried out when there is the possibility of employing breast-feeding. From previous considerations it is evident that even this must be used with the greatest care, and must be limited to a very brief period on the breast at four hour intervals or, in case the sucking reflex is not present, to a few teaspoonfuls of expressed breast-milk at the same intervals. If the first critical days are passed and if the food is retained, without causing renewed vomiting or diarrhœa, then the battle is half won, provided the advance is made slowly. The power of sucking soon returns, and there comes the happy satisfaction of observing a complete change of such patients in a short time, with rapid gain in weight, caused particularly by the great retention of water in the tissues. The rosy color of the skin returns, the features regain their infantile

softness, the patients suddenly begin to pass an increased amount of a light colored urine containing daily less albumin, and their movements gradually assume the characteristics of the normal breast-milk stools. The treatment must be supplemented by the most careful attention to the skin and accessible mucous membranes, which means frequent changing of napkins, thorough cleansing of the anal region with cotton and vaseline, frequent cleansing of the mouth with 3 per cent. boric acid or 0.2 per cent. corrosive sublimate solution, baths and so forth. Care must be taken to supply plenty of fresh air. This method of treatment is also applicable to children at the end of their first year.

The results are much more precarious when there is no breast-milk at our disposal. The ease with which acute attacks are brought on by concentrated animal milk necessitates an especially slow advance, it also requires care for the quality of the product employed, and the closest supervision of the cleanliness of the bottle, the nipple, and of the quantities given and the intervals employed. Our task becomes all the more difficult because the reawakened feeling of hunger results in a marked increase in the nutritive requirements of the child. At this stage the milk must be very much diluted, and it is still better to begin with the administration, two or three times a day, of some cereal water, made from rice flour, oatmeal, or barley flour, to which milk is gradually added, and the strength of the food is only increased when the movements have been normal for several days, and when the daily amount of urine has risen. The quantity given at each feeding, and the total daily quantity must remain much less than the requirements of normal children of the same age, on account of the low weight and greatly lessened digestive power of these patients. The drugs mentioned above, which improve the digestive power of the stomach, such as pepsin, hydrochloric acid and so forth, find their place here; also the so-called intestinal antiseptics, of which the antiseptic value is theoretical rather than based on fact, but which empirically seem to supplement the improvement in the movements caused by the careful regulation of the food. I have seen no effect from the highly recommended use of lactic acid, although I was not able to get the children to take it in the 5 per cent. solution proposed by Hayem and Lesage as the minimum. On the other hand, bismuth salicylate in a 1–5 per cent. emulsion, with 10 parts of glycerin in 100 added to disguise the taste, beta-naphthol in the same strength, bismuth subnitrate best given in powder, .02-.03 gram ($\frac{1}{3}$ to $\frac{1}{2}$ grain) 3 or 4 times a day, and similar drugs, are of decided value. One must always be on the watch to observe any change in the movements, especially an increase in the number, or the appearance of a bad odor, mucus, or curds. In such a case the strength and quantity of the food must be reduced, and perhaps a cereal water given again or even a day of starvation may be advisable.

Children overfed with cow's milk otherwise properly obtained and prepared, in the later months of infancy frequently present symptoms pointing to incomplete digestion of the food and to *bacterial fermentation* in the large intestines with its resulting irritative symptoms. Their gain in weight begins to stop, their good color changes to pallor, there is slight fever. The number of movements increases, their odor becomes rather foul suggesting bad eggs, their appearance deviates from the normal in that they show an abundant admixture of mucus, which appears at first in the form of light colored lumps scattered through the otherwise homogeneous pulpy mass. If these symptoms are not heeded and if no attempt is made to relieve them through the management of the diet, they continue till a sharp rise of temperature and a true diarrhœa rather suddenly develop. The character of the movements and their method of evacuation point to the colon as the chief seat of the disturbance. The mucus in the movements increases to such an extent that it becomes evident to those in charge of the child, the odor becomes constantly fouler, and evacuation causes pain and marked tenesmus, which manifests itself in a prolapse of the greatly reddened and swollen mucous membrane of the anus which is forced out with each movement and is gradually drawn back. Finally, fecal movements alternate with others consisting only of little lumps of gray or grayish red mucus, which later are more like pus and are frequently streaked with blood or markedly blood-stained. These movements preponderate more and more and become constantly more numerous, up to 20 or 30 a day. Meanwhile the fever continues, the pallor increases, the general condition suggests a severe illness, the weight shows a corresponding marked fall, the region around the anus is excoriated by the constant irritation of the frequent movements, and the anus is in a permanently spastic condition. If the illness is further neglected, there can develop, in the first place, an infection of the urinary tract occurring especially easily in girls, which leads to cystitis, and to processes ascending from the bladder toward the kidneys. In the second place there can develop those infectious complications of the skin and internal organs, to which every severely sick infant is easily liable. We have before us the picture of an inflammation of the large intestine. If the milk taken is bacteriologically pure, and if there is no other opportunity for infection, such as there is in institutions where such processes occur from a contact infection, then occurrence of these cases is strongly in favor of Biedert's view of injurious food residue as the cause. This author perceives in the high casein content of cow's milk its chief injurious quality, and assumes that this proteid being insufficiently digested, remains stagnant in the large intestine where it undergoes the decomposing action of the intestinal bacteria, and exercises a local irritation on the mucous membrane. This view has recently been contested by

Heubner and Czerny-Keller on the basis of absorption experiments, and of the unreliability of the method of estimating the proteid in the discharges. Nevertheless just such cases as were described above make it very probable that the local effect of this residue of cow's milk is of importance, and the results of treatment favor this view.

Treatment can consist in the combination of dietetic measures with medicinal or mechanical measures, or with both. Milk feeding is suppressed and instead a cereal decoction is given. In order to remove the fermented contents from the intestine a mild unirritating purge can be used. Calomel is out of place in such cases, but one can give one of the following:—castor oil, one or two teaspoonfuls Hufeland's powder (powdered magnesia with rhubarb), as much as can be held on the point of a knife, in tea or boiled water; Kurella's powder (compound liquorice powder), in the same doses as Hufeland's; or a mixture of equal parts of tincture of rhubarb and hydromel infantum, a teaspoonful every two hours till effective. This treatment is continued until the appearance of a fecal movement and is followed by relaxation of the tenesmus and diminution of the excretion of mucus, as the cereal-water diet prevents decomposition in the large intestine and so has an indirect action upon the inflamed mucous membrane. Frequently however this treatment is insufficient and requires to be supplemented by local treatment, the object of which is to remove the masses stagnant in the large intestine, to wash the mucous membrane clean, and possibly, if the fluid employed is slightly astringent, to reduce the hyperæmia. If one simply wishes to evacuate the intestinal contents, irrigation with boiled water is sufficient. If there is a high temperature, the irrigation fluid is used cold. It is introduced by means of a Nélaton catheter, which is pushed up as high as possible, and then slowly withdrawn, in order to irrigate all parts of the mucous membrane within reach. According to the age and development of the child from $\frac{1}{2}$ to 1 litre is allowed to run in, under the slight pressure of about $\frac{3}{4}$ to 1 metre (2 to 3 feet) of elevation. For antiseptic irrigation which can be employed either in the first place or following the irrigation with water, 2–$2\frac{1}{2}$ per cent. boric acid solution, or a solution of salicylic acid and thymol, 1–$1\frac{1}{2}$ per cent. of each, may be used. Where an astringent action is desired I use almost exclusively the liquor Burowii ("aluminum acetate solution") diluted and filtered (alum 5., plumbi acetat. bas. sol. 25., aq. destillat. 1000.), or a solution of alum $\frac{1}{4}$ to $\frac{1}{2}$ per cent. As to irrigation with tannic acid, I have seen cases of collapse come on after the use of fairly weak solutions (0.5–1 per cent.), and consequently I do not use this drug any more. In acute cases with severe symptoms one should irrigate several times a day, and should not discontinue irrigation until the tenesmus has disappeared, and the mucus in the discharge is reduced to a minimum, which usually takes

place in three or four days. Not until after the lapse of this period is it advisable to begin adding milk to the food in gradually increasing quantities, and in addition one may then use such drugs as tannigen (3 to 7 grains three times daily), tannalbin, in like dosage and bismuth (the amount easily held on a knife-point).

The course of this process in the artificially nourished is in general more favorable than that of those diseases which also involve the stomach and the small intestine. Nevertheless it may happen that inflammation localized in the beginning in the large intestine, can follow an ascending course to the stomach. On the other hand cases which begin with convulsions, severe prostration, and high fever may either be fatal in the acute stage, usually through pulmonary complications, or they may, after the passing of the stormy symptoms, result in a condition of chronic ill-health which may continue for weeks or months. The boundary lines between these inflammations of the large intestine and true dysentery do not appear to be at all sharp, especially as recent progress in bacteriology has resulted in the demonstration of the same exciting organism in both forms of disease. The organism is also to be found in a considerable percentage of cases in the intestinal contents of normal infants, and probably in the conditions described above may pass over from a condition of latent presence into one of manifest infection.

All the forms of diseases mentioned up to this point can find their outcome in the symptom-complex of the so-called *Atrophy.* This condition can develop as a result of overfeeding with cow's milk, without any very noticeable disturbances of digestion, although always preceeded to a slight extent by such disturbances. The greater burden which is laid upon digestion by even the proper use of cow's milk in rationally fed infants, leads to exhaustion of the digestive function if the quantity of food given surpasses the proper average. In what way these disturbances are caused, whether they are due to insufficient absorption resulting from anatomic injury to the absorption apparatus, or to disturbances of the intermediate metabolism particularly from diminution of the oxidizing function of the liver, or to a condition of intoxication from the formation of abnormal products of metabolism, or to withdrawal of alkali and consecutive encroachment upon certain neutralizing bodies, or to a combination of several of these factors is still unknown, and will be thoroughly discussed in another part of this book. What we observe clinically is that after a rather long period of stationary or irregularly fluctuating weight-curve, a progressive loss in weight occurs. At the same time there is increasing pallor of a light yellow color, or of a tint inclining toward a lead-gray (the latter especially in very young infants), slight enlargement of the peripheral lymph-nodes, and marked prominence of the abdomen. The loss of

weight finally leads to complete disappearance of the fatty tissue, and to the most extreme emaciation. The cranial bones appear to be overlapping, on account of diminution of the volume of the brain and partial disappearance of the cerebrospinal fluid, and to be notably hard. The fontanelle is small and depressed, the face presents a senile expression, the eyes are sunken in their sockets and surrounded by bluish circles, and the neck is long and thin, the sternomastoids, larynx and jugular depressions being sharply defined. The chest is narrow, flaring suddenly in its lower portion, the supraclavicular depressions are deep, and every rib can be counted. The abdomen is tympanitic and distended, rising above the level of the chest, and forms the only contrast with the skeleton-like condition of the rest of the body. All this presents a characteristic picture, as is strikingly shown in the photographs

FIG. 18.

Advanced atrophy in a child five and a half years old.

of the two cases of this kind, from the Gratz Children's Clinic and the Dresden Infants' Home (Figs. 18 and 19). Also, the hernia so frequent in this condition is to be seen in one of the pictures. The extremities look like specimens preserved in alcohol, the skin hangs loosely over them, and the thighs especially appear to be stuck in bags of skin much too large for them. The spines of the vertebræ stand out sharply, and the outlines of the shoulder-blades, the sacrum, and the pelvic bones are sharply marked under their covering of thin, dry, parchment-like skin. Everything is sharp and angular, and nothing remains of the softness and roundness of form which characterizes healthy infants. The visible mucous membrane appears dry, the tongue is coated, the conjunctival secretion is slimy, and the skin is the seat of pustules, papules, and ulcers, which, particularly in the anal region, can develop into wide-spreading and deep-reaching inflammations. The sub-

cutaneous tissue is often studded with abscesses of varying depth, and of a size from that of a pea to that of the clenched fist, which can become confluent and form irregular pus cavities of wide extent. This pitiable little being shows plenty of appetite and takes the bottle eagerly. Vomiting is rare, and the movements are apparently not bad, only very small, pale, with lumps of mucus and a putrid odor. The urine is usually abundant and decomposes rapidly, so that the ammoniacal odor together with the stench of the movements makes the examination and nursing of such children extremely disagreeable. The condition is one of progressive cachexia, coming on after preliminary disturbances of digestion, abnormalities of assimilation, and periods in which the metabolic equilibrium is retained. Such patients are very unprotected objects of attack for secondary infections, appearing as suppurations in various organs, gangrenous processes, pneumonic areas, and so forth, which cut short the course of the process. Or acute attacks of vomiting and diarrhœa may rapidly end the scene. When these terminations do not occur, the process, through insufficient utilization of the nutriment given and the consequent melting away of the body substance, leads to an extreme degree of emaciation.

Fig. 19.

Advanced atrophy with congenital hernia in a ten-months-old child.

This outcome of a disease of nutrition is especially unfavorable, because, in the first place, its cure constitutes one of the most difficult of therapeutic tasks and requires the greatest patience; in the second place, the survival of momentary danger does not mean that the battle is won, since such children frequently remain injured for years and appear to be very little resistant toward intercurrent diseases.

In advanced stages of the disease a partial prospect of rescue is offered only by breast-feeding, or rather by the use of human milk

drawn artificially from the breast until the power of active sucking is re-established. As Keller has recently shown, if this treatment is persisted in, and if a period of initial loss and prolonged stationary weight be survived, then, as a reward for our trouble, there finally occurs a fundamental alteration of the digestive power, and a gradual though steady gain in weight. The healthy color of the skin returns, the wrinkles and sharp angles are lost, and the child becomes lively, notices its surroundings, and seems to enjoy life. Nevertheless, many weeks often elapse before this end is attained, and the most energetic supervision is required on the part of the physician over those in charge of the child, in order to prevent them from losing patience and from going back to dangerous experimentation with the feeding.

Other measures than breast-feeding are so uncertain and subject to accident, that in every fresh case one is compelled to try experiments of which the results are always doubtful. The instituting of artificial feeding, if no wet-nurse is obtainable, is best begun with a period of starvation lasting from 24 to 36 hours. Nobécourt has recently recommended physiologic salt solution instead of the usual water or tea, because its use is said to render the loss of weight much less. The experiments of Méry, whose treatment has been more completely described by P. Lesage, seem to me especially interesting. He gave a vegetable soup over a long period, for a week or longer, which was eagerly taken by the children. It caused a gain in weight, probably from retention of water, and a fundamental alteration in the function and flora of the intestine. Finally, proper nutrition is re-established by the gradual addition of milk. The method of preparing this vegetable soup is as follows: 60 grams (2 ounces) of potatoes, 45 grams ($1\frac{1}{2}$ ounces) of carrots, 15 grams ($\frac{1}{2}$ ounce) of turnips, and 6 grams ($1\frac{1}{2}$ drams) each of dried peas and beans are put into a litre of cold water, and boiled four hours in an earthenware or porcelain vessel. The fluid is then poured off and its amount raised to one litre by the addition of boiled water, and 5 grams (1 rounded teaspoonful) of salt are added. A yellowish, slightly cloudy fluid of pleasant taste is obtained. It must be prepared at least once a day, in hot weather twice a day, and must be kept in a cool place, preferably upon ice.

Heubner recommends that after a preliminary resting of the bowel a fundamental change in the food be made, and in cases like those under consideration where the cause is overfeeding with cow's milk, he advises a purely carbohydrate diet, to which milk is gradually and carefully added after complete alteration of the discharges has taken place. In very desperate cases, and these are fairly common, raw cow's milk can be tried. Its mode of action is not yet clear, and does not consist in the suggested supplying of ferments, but at times, as I can confirm from experience, it produces good results. Frequently, in spite of every

effort, no food can be found suitable to the requirements. Buttermilk and cereals especially, are often entirely useless in such cases. Every change of food leads to acute exacerbations with severe vomiting and diarrhœa, cooling of the body, and collapse, which render all results entirely questionable. Or else, if these "catastrophes" (Heubner) are avoided, the loss of weight constantly progresses, the emaciation advances further, secondary infection appears, and the illness cannot be halted.

The very fact that such conditions of atrophy come on in breast-fed infants only as a result of a septic infection with a consequent widespread parenchymatous degeneration of the organs of assimilation, and not as a disease of digestion, and the fact that breast-milk alone is able with apparent certainty to bring about a complete cure of such cases, are evidence of the immense superiority of this method of nutrition, and of the great danger of underrating its importance for proper development.

Then, as we have already mentioned, if we obtain a gain in weight and an improvement of the gastro-intestinal function after long trying and much trouble, nevertheless not everything is yet won. Severe anæmia may persist for a long time, and frequently the processes of repair are so imcomplete, that, on the basis of a foregoing disease of nutrition, a disease of the blood (most often anæmia pseudoleukæmica infantum) may develop, to which the infant finally succumbs. Or else, an infectious disease is acquired later, in which the severe and often fatal course is evidence of the lessened resistance of an organism injured by former illness.

For these reasons, even when permanent gain and improved digestion are attained, treatment has not reached the limit of its utility. It must direct its attention to the secondary infections which often flare up in the strengthened body with renewed severity, and it must be directed toward relieving them and preventing their further spreading, by the most careful cleanliness, by evacuation of pus, by the shutting off of discharging wounds from their surroundings, and by similar measures. Apart from this, treatment must see to the improvement of the conditions of life by supplying an abundance of fresh air. It must contend against anæmia with preparations of iron, such as ferri carbonat. sacch. 0.1 gram (1½ grains) 2–3 times a day, or ferri citrat. in the same doses or of the various newer iron preparations such as anæmin, hæmatogen, Drees' albuminate of iron, hæmaticum, etc., in teaspoonful doses. Comby and Narbel recommend the injection of Clin's lecithin into the muscles of the thigh, 1 c.c. (♏ 15) daily; but I have had no personal experience with this drug. The treatment in this second stage must also include avoidance of opportunities for infection. A stay at the sea-shore or in a mild mountain climate will also possibly favor the return of physical strength.

B. **From Insufficient Food (too great dilution).**—This cause of the diseases of nutrition, which is not rare in breast-feeding, is only seen to occur in artificial feeding in one way. A food of too little concentration, such as too highly diluted milk, is given, with which the infant, though taking an abundance of fluid, is nevertheless unable to satisfy its caloric needs. This type of illness has recently diminished in frequency, because, under the influence of Escherich, Soxhlet, Heubner-Hofmann and others, the use of smaller volumes and higher concentrations of food in connection with partial replacement of the fat by carbohydrate, has been made popular, especially in Germany. It is found more in countries which are more unaccustomed to artificial feeding, and where the mothers instinctively dilute the cow's milk very greatly with water or tea, in order to favor the infantile digestive organs. In such cases there is a combination of the symptoms of overloading with fluid with those of starvation, which manifests itself clinically in the appearance of profuse diuresis (especially in dilution with tea), and of infrequent small movements and sinking in of the abdomen. If these premises are borne in mind, the results follow naturally. A relatively large amount of fluid, up to $1\frac{1}{2}$ or 2 litres in 24 hours, is taken, of which the food content does not satisfy the requirements of the child, On the other hand, this great overloading with fluid can lead to dilatation of the stomach with resulting retarded digestion, and as a further result of the damage to digestion, occur loss of appetite, infection of the gastro-intestinal contents, and the various other effects connected therewith that were described in the former chapter.

The **treatment** of these conditions consists in marked restriction of the amount of each feeding, while at the same time its nutritive content is increased. This can be attained preferably by adding cane or beet-sugar and gruels rather than by adding a milk-sugar solution of high percentage. The dilution of the milk should not be under 50 per cent. The total quantity taken in 24 hours should, except in the first days of life, amount to about 15 per cent. of the body weight (Epstein), or 50–200 c.c. per kilo (Biedert). These amounts should only be retained if the digestion and general appearance of the child improve, and if the weight maintains a steady increase. The possible injuries resulting from overloading with fluid, such as gastrectasis, vomiting, and so forth, must be treated in accordance with the principles developed above.

C. **Through Failure of Utilization of the Food (Either as a Whole, or in its Individual Constituents).**—The researches of the last few years on the significance of the individual peculiarity of the proteids of various kinds of milk, the most important results of which I have already mentioned, have been invoked in explanation of the intolerance of cow's milk noticed in many cases. It is not necessary to settle the controversy here, whether the results are due to the action of the cow's

milk as a whole, or to that of its proteids. We have only to point out that there are children who, when given the very smallest quantities of cow's milk, react with the severest symptoms, such as fever, violent vomiting and diarrhœa, convulsions, toxic erythemata, and so forth. The appearance of a leucocytosis in their blood, and the symptoms of increased excitability of their central and peripheral nervous systems are further evidences of the intense reaction of their organism to the foreign nutriment. Which constituent of the milk is ultimately to be blamed is still undecided, although the fact that repeated observations in older children showed a similar intolerance toward hen's eggs is very much in favor of the toxic influence of the foreign albumin. The practical conclusions to be drawn from such observations are, that whenever any such stormy reaction arises either in infants fed with cow's milk in the early weeks, or in children given cow's milk as part of their nutriment in the normal weaning period, we must go back at once to the breast. It is obviously possible to try immunization against the foreign proteid, and this can be attempted by the use of gradually increasing amounts of the particular kind of milk either by the mouth or by subcutaneous injections. The former method according to Finkelstein, in many especially sensitive children does not attain the desired end, since every administration of even the smallest quantities of cow's milk is attended by the most severe and sometimes even dangerous symptoms. The second method, subcutaneous injections of sterile cow's milk, has only been tried in animal experiments. On the other hand, Schlossmann, on account of the impracticability of milk injections, has tried *injections of cow's serum.* The reports of his observations sound very encouraging, as by this process he was apparently successful in immunizing the child rapidly and permanently against cow's milk proteid, without any notable reaction. I will not build any castles in the air on this subject, particularly as Schlossmann's conclusions have encountered strong opposition on the part of Finkelstein. They only point out the lines on which further progress may be made. At first 0.1 c.c. of cow's serum is injected, and the injections are repeated in increasing amounts (up to 0.5 and 1.0 c.c.) after the reaction following each has subsided. The reaction consists in fever, acceleration of the pulse and respiration, pallor of the face, and at times also there is an erythematous or urticarial rash. The injections are continued until there is no longer any reaction, at which time permanent immunity is obtained. I believe it is going too far, to designate such a proceeding as a routine method and to undertake it as an act properly accompanying artificial feeding or weaning. Nevertheless in cases in which the first administration of cow's milk starts up severe symptoms of reaction, an attempt at immunization with cow's serum is always in place.

I have already had occasion to refer to one probable injury from

excess of proteid in cow's milk, when speaking of Biedert's view of injurious food residue, which finds its particular clinical expression in the appearance of irritative symptoms localized primarily in the large intestine. This "*proteid injury,*" to accept the very appropriate term recently introduced by Czerny and Keller, has been strongly denied, although no very convincing proof has been brought forward against it. It is a fact easy to confirm by observation, that when strong dilutions of cow's milk, or whole milk are given too early, diarrhœal movements not infrequently develop, of which the number, excess of mucus, odor of foul albumin, and passage with tenesmus indicate a strong probability that incompletely digested proteid bodies are stagnating in the lower bowel. These form a favorable field for putrefactive organisms, and the resulting products of decomposition exercise an irritative action which is at first local, but which in its further progress can form the starting point of severe symptoms, ascending and involving the entire intestine and the entire organism of the child. The recognition of the excess of proteid in the movements cannot be made with certainty, as the Millon's reaction, which is supposed to do this, loses considerable value, because it can also be produced by a marked increase in the number of intestinal bacteria present. Clinical experiences, especially the rapid effectiveness of discontinuing milk and substituting cereal water in these cases are in my opinion strongly in favor of Biedert's assumption, and of the authorization for the view of proteid injuries.

Czerny and Keller have described disease pictures, which they attribute to the unsuitability and injurious results of the fat of cow's milk, tolerated especially badly in these cases. They call this "milk injuries," though I believe that logically, "fat-injuries," or the somewhat ponderous term "*cow's-milk fat-injuries,*" would be more correct. The first symptoms in this disturbance consist in poor sleep, restlessness, and the passage of gray to white discharges, constantly becoming drier, and with a plainly fecal odor. In its further progress appear pallor, loss of firmness of the flesh, and increasing distention of the abdomen. The hard light gray movements consist mainly of insoluble fatty soaps, their color is to be attributed to abnormal process of reduction in the intestine, through which the bilirubin is converted into colorless urobilinogen (Langstein). In a still more advanced stage there is weakening of the abdominal wall, and the occurrence of secondary infectious processes and heart weakness, which manifests itself in a grayish blue color of the skin, and muffling of the heart sounds, particularly the former. Treatment of this condition can be attempted either by reduction of the amount of food, which only helps in the initial stages, or by dilution of the milk with the addition of carbohydrates. Czerny and Keller attain the latter end in the milder cases by adding a cereal decoction, and in the severer cases by a food which consists

of diluted milk with the addition of two carbohydrates, a sugar and a starch. The indication for the use of two carbohydrates is found in those cases in which the giving of milk diluted with a cereal broth is not followed by gain in weight and change in the appearance, color, and consistency of the movements. Of these food mixtures the barley soup proposed by Keller, a modification of Liebig's soup, and buttermilk have stood the test, of which the method of preparation is described in another part of this book. In my clinic we have had in a rather large number of cases good results with the preserved buttermilk proposed by Biedert and Selter, and prepared in the dairies at Zwingenberg in Hesse. This food has the advantage of a simple method of preparation, in that it is merely diluted with three to five volumes of lukewarm water according to the age of the child, but it is rather expensive. The well-known bad digestion of starch in the first months of life makes breast-feeding, or at any rate feeding with the preparations mentioned above which do not contain starch, appear the most desirable methods of dietetic treatment, while, from the age of four months on, barley soup and buttermilk can find their use. In the subsequent treatment, after the appearance of a regular gain in weight and good movements, which takes a varying length of time to occur,—usually from four to six weeks,—the strength of the milk in the barley water is raised, or feedings of cow's milk are substituted for single buttermilk feedings, until finally a pure milk diet is obtained.

Beside this "fat-constipation," there is also a "*fat-diarrhœa*," coming on from the giving of an amount of fat which is excessive for the particular child. This has been described by Demme and Biedert, and manifests itself in the appearance of loose, pale-yellow, foul-smelling, oily glistening stools, in which beside fat-crystals (clusters and needles) can be demonstrated microscopically also fluid fat in the shape of drops and pools. It is not exclusively to be observed in breast-fed infants, but is found also at times in the artificially fed, chiefly from the giving of milk mixtures with too abundant fat, as in mixtures made with milks which yield an excess of cream, or in cream mixtures. It can also be produced artificially when one increases the fat in the food of children who are suffering from constipation. Treatment of this condition is obviously to be instituted by reducing the amount of fat in the food, perhaps through stopping the addition of fat, or, in case this does not suffice, through proper dilution of the milk with additional carbohydrates in the form prescribed above.

It happens often enough, that with failure to observe the first stages of this form of disease, there develop imperceptibly those results which we know under the designation of atrophy, the treatment of which causes the greatest trouble, and can often only be successful if there is a return to breast-feeding.

Also the course of this process can be interrupted by severer and more acute symptoms of irritation referable to the gastro-intestinal tract, which manifest themselves in more frequent vomiting and in loose movements with mucus, epithelium, and leucocytes. This often leads unexpectedly to a rapidly fatal termination, and consequently its appearance requires the greatest attention, and immediate energetic treatment according to the rules developed above. Finally secondary infections are to be thought of, which easily attack such patients, especially in the later stages of their disturbance. These may result in threatening complications in the form of suppurations, or inflammatory ulcerative processes in the region of the skin, as well as diseases of the respiratory organs such as capillary bronchitis, and lobar or lobular pneumonia.

The bad custom still prevalent in many countries of feeding children from the first weeks of life with cereal decoctions, leads also to a number of disturbances of nutrition, which Czerny and Keller designate by the name "*starch-injuries.*" The widely increasing manufacture and advertisement of cereal infant foods contributes not a little toward this custom. The disturbance may also be caused by giving children a food of which the preponderating constituent is carbohydrate in the form of soup or gruel made from flour. In Prague we have little opportunity to observe this disturbance in its pure form, since our infants are almost exclusively kept at the breast in the early months of life, and are only at a later period fed with preparations containing carbohydrates, usually groats, zwieback, and crackers. From this I have gained the impression that this mixed diet, though certainly not very rational and usually also entirely superfluous, is nevertheless not followed by any especially injurious results. Beside intercurrent acute disturbances of a mild character, such as foul slimy diarrhœa which quickly ceases when the supplementary feeding is stopped, no grave disturbance and particularly no chronic diseases of nutrition develop. Heretofore I have been inclined to attribute to this proceeding, which is almost universal here, an influence contributing to the rather severe form of rachitis very prevalent in Prague, but this is easier to surmise than to prove.

The Breslau authors, whose book I often find occasion to cite, have at their disposal in this particular a much greater material for observation. I can draw upon my own experience only for the cases of this kind often seen during my service as assistant in Munich, the course of which, as far as my memory serves me, agreed with the description given by them. Consequently I will repeat their description here in its essential particulars. A few children show no visible injury from an exclusive or preponderating cereal diet, this being due to individual differences in their supply of digestive ferments. Apart from these, most children show somewhat moist movements, varying from a bile

stained to a dark brown color. In the younger children especially, or if there is a very abundant administration of cereals, the movements contain undigested starch, which is to be recognized microscopically and chemically in the way described above. Their reaction is acid, and the odor is faint if there is not much mucus passed, whereas with more pronounced fermentation it is pungent or resembling acetic acid, and with a larger admixture of mucus it is fecal. In the beginning such children look very well, are well nourished, with firm muscles, and slightly prominent abdomen, and in respect to good humor, liveliness, and interest in their surroundings behave in a fairly normal way. An important sign that the condition is growing worse is the hypertonicity of the muscles described by Gregor, with which is associated increase of the electric excitability, this subjects the children to the dangers of eclampsia and spasm of the glottis. The weight curve, favorable in the very beginning, soon shows a prolonged stationary condition, and finally a progressive loss, which can lead to the most pronounced atrophic condition. When diarrhœal movements appear, the weight falls rapidly, and children are liable to such intercurrent catastrophies. Still more often there are secondary infections, which waylay the well-nigh unprotected organism.

With progressive or continuing injury the good appearance disappears to be replaced by pallor, the firmness of the flesh diminishes, and skin and muscles have a peculiar doughy feeling, without true œdema being present.

The treatment has for its first object excluding the starch from the food and replacing it with proteid and fat. The surest way of attaining this end is by putting such children again to the breast, whereby bacterial injuries at least are practically excluded, and a variety of fat is offered which can be most easily utilized by patients severely injured in their assimilating power. Even with a wet-nurse they recover themselves but slowly, and only after rather long and constant gain in weight may a careful trial of giving them starch be made. This is best done in the form of milk diluted with a cereal decoction. The improvement of the general appearance and the disappearance of the hypertonicity give evidence of the favorable influence of breast-feeding even before the appearance of a gain in weigh

When no wet-nurse can be obtained, cow's milk as nearly pure bacteriologically as possible is given, which for young children, under three months, is diluted with whey, which can be prepared at home by coagulating out the curd with rennet. Dilutions of cream and condensed creams have the disadvantage that with their use constipation with fatty soaps in the movements frequently develops, which requires alteration of the treatment, or else frequent vomiting makes a good result questionable.

D. **From Bacterial Contamination of the Food.**—How seldom, and under how great difficulties we are able to obtain and keep a bacteriologically clean milk, I have already explained. It is easy to understand, that at the time when we first become informed as to the sources of contamination of cow's milk and as to its great content of bacteria constantly increasing under certain conditions, we considered the riddle of the etiology of gastro-intestinal disease in artificially fed children to be solved, and the way toward their cure to be pointed out. Since then statistics and daily experience have taught us that a condition of freedom from bacteria attained and preserved by sterilization or in any other way can only remove a very small part of the evil. A large number of injurious factors remain, whose prophylaxis and treatment must be sought in an entirely different direction. The question now is, whether there are any disease pictures of which the causal connection with markedly contaminated milk is established. Our results on this subject are not very great. The number of such observations becomes still more limited, if we demand exact proof of the causal connection, such as the recognition of the supposed exciting agents in the milk and movements of the children, and if we only recognize those infections which are caused by organisms not normally present or at most rare in the intestinal contents. Certain it is that this subject has long been insufficiently worked out. In countries where there is much artificial feeding there is observed every summer a tremendous increase of acute diarrhœas, which present in their course the picture of a severe infection, which usually develops during apparently perfect health, or on a basis of unimportant disturbances of digestion. These cases point with compelling force toward food contamination as a factor.

It appears from the work of recent years to be established with constantly greater certainty, that *streptococci* play an important part in the causation of acute and chronic gastro-intestinal infections, which occur as the result of the taking of cow's milk contaminated with bacteria. Lesage and Thiercelin, Nobécourt, Escherich and his pupils, Finkelstein, and others, as well as more recently Petruschky and Kriebel mention the frequency of finding streptococci in the discharges of artificially nourished infants with gastro-enteric disease, and also the demonstration of these organisms in badly kept cow's milk. They gain entrance into the milk from inflammatory processes in the udders of the animals, and also from the dirt of the stable, the manure, and the hair of the animals, etc. They can, as in the observations made by Escherich, form the only exciting causes, in which case there is produced the clinical picture of a severe disease localized primarily in the large intestine, which either ascends from there and leads to general symptoms, or breaking through the intestinal wall, produces general infection through the blood-and lymph-channels. This disease is ushered in

by fever and prostration, and numerous mucous or mucopurulent and blood-containing discharges, which contain as predominating microorganisms the Gram-staining cocci in long chains or in little groups and clumps. Convulsions follow, and increase of the intestinal symptoms, the appearance of algidity, and collapse can terminate the scene in a short time. When it is a case of invasion of the lymph- and blood-channels proceeding from the intestine, the onset is less stormy, the fever is at first only slight, and the movements have a more serous character, pointing to the seat of the lesions in the small intestine. The movements soon change, in that their increasing content of mucus, pus, and blood points to an extension of the inflammation into the large intestine. Then follow cystitis, bronchopneumonic symptoms, and otitis, which are the cause of death. This occurs either in coma, or from the sudden appearance of collapse. The demonstration of the exciting organisms in the urine, blood, and lumbar puncture fluid of the living child is evidence of the general infection of the body. Fleeting or more prolonged affections of the skin, usually manifesting themselves as polymorphous erythemata, hæmorrhages into the skin and visible mucous membranes, symptoms of meningeal irritation, suppurative inflammations of the serous membranes, are signs of the severity and diverse localization of the infection. The diagnosis of such infections is based on the characteristic bacteriologic picture, on microscopic examination of the stools, and the possible demonstration of the same organisms in the urine, blood, and cerebrospinal fluid. The marked affinity of these streptococci for the organism can be demonstrated by the specific agglutination. Specific treatment, by means of the various antistreptococcus sera has given no results, and therefore the symptoms must be combated by water diet, intestinal irrigation, careful nursing, and so forth, as described above. In addition, on account of the contagious character of the disease, evidence of which has been frequently brought forward, care must be taken as to isolation of the cases and proper disinfection of the linen, discharges, and so forth.

Infections with the *bacillus pyocyaneus*, another organism not present in the intestine of the normal infant, have likewise been repeatedly observed. (I cite the observations on infants of Williams and Cameron, Kossel, Lesage, Thiercelin, and H. Meunier, quoted by Nobécourt, together with the cases of Escherich and of Nobécourt himself.) The clinical picture of this infection is by no means sharply distinct. Sometimes it is that of a primary disease of the intestine with secondary toxic symptoms, which comes on with a sudden rise in temperature, tympanites, severe involvement of the entire organism, and the appearance of bright green discharges, in which the preponderance of the pyocyaneus can be observed. At other times, it is rather a localized affection of the large intestine with stools full of mucus and showing

a variety of blue and green colors, which also contain the same micro-organisms as the predominant variety. General infection does not occur, or at any rate the examination of the blood and other fluids have so far given negative results. For this reason the severe general symptoms are attributed to the formation of toxines.

Proteus infection (cited by Nobécourt, who mentions cases of Booker, Lesage, Brudzinsky, Tobiesen, and their observations), appears with a varying disease picture, at times as a cholera infantum of very acute course, at times as chronic intestinal disease with a tendency toward atrophy, at times as a simple dyspepsia of mild clinical course. Its diagnosis rests upon the demonstration of the exciting organisms in marked preponderance in the stools, and its treatment follows the usual rules.

Duval and Basset were the first to point out the significance of the *dysentery bacillus* in a number of severe gastro-intestinal diseases. The results of a joint investigation carried out in America have been collected by Flexner and Holt. They have been partly confirmed by Leiner and Jehle, and show that this disease can occur as an acute or subacute primary infection, and also as a secondary infection. The clinical symptoms can present all variations from the mildest to the severest gastro-intestinal manifestations. The stools are characterized by an admixture of mucus, and the bacilli are particularly to be demonstrated in these mucous portions. The commonest clinical type of the disease is a catarrh of the large intestine, with numerous muco-purulent and bloody movements, with tenesmus, and with an acute febrile onset. The treatment consists in water diet and prophylactic measures to prevent contact infection. Serum treatment has been employed without apparent result.

Finally, there is still to be considered the bacterium coli, which for a long time has stood in the foreground among the suspected causes of infection. Further results of researches have shown that this ubiquitous organism, by its favored development on the usual culture media easily overgrows other varieties of greater etiologic importance, and that the facts adduced in support of its pathogenicity, such as virulence in animals, agglutination, and so forth, do not wholly bear strict criticism.

As to the etiologic rôle of the anaërobic intestinal bacteria, we must await further investigation.

An observation frequently made is the appearance of an epidemic of gastro-enteric cases, and cases of enteritis, particularly in institutions, and the demonstration of contact infection in such cases. At times the infection is communicated by the hands of the nurses, to which infectious material clings, at times by the nipples of the bottles, or it is carried from the anus by napkins and thermometers. The knowledge

of such occurrences has led to stricter supervision of the nursing in institutions, and has resulted in isolation of the children, careful asepsis and thorough disinfection of the implements used. The measures of Finkelstein in the Berlin Municipal Orphan Asylum represent the highest perfection of such care. Their result proves that beside digestive causes and contamination of food still other factors are involved, of which the prevention has a significant influence upon the figures showing the frequency of disease and mortality.

LOCAL DISEASES OF THE STOMACH AND INTESTINES IN EARLIEST CHILDHOOD

BY
PROFESSOR R. FISCHL, OF PRAGUE

TRANSLATED BY
DR. J. H. MASON KNOX, JR., BALTIMORE, MD.

1. ULCER OF THE STOMACH AND DUODENUM

THIS affection is extraordinarily rare in childhood and particularly so in infancy. It offers great difficulties in *clinical diagnosis*, when, as is frequently the case, especially in round ulcer in this period of life, bloody vomiting, and stools containing blood which has its origin in the upper bowel, are both lacking. Moreover, the vague, indefinite pain, not always localized in the gastric region, as well as the vomiting of the food and progressive loss of weight, are considered to be due to some process in the lower abdomen, as for instance appendicitis. On this account most of the cases reported (these have been collected by Rehn and Bechthold), were not diagnosed during life; indeed, in the instances observed by Bechthold, the condition was not recognized after laparotomy and was first discovered at autopsy. The small amount of hydrochloric acid, produced in the infant's stomach, the rarity of hyperacidity at this age, the rapid emptying of the food contents into the small intestine, and the absence of mechanical disturbances, render the relative *scarcity of cases* readily understood. Without claiming to have made an exhaustive examination, I was able to find only twenty-one cases, the youngest of which was a child of two and one-half months.

The **course** of the disease was but seldom so typical that the clinical diagnosis could be made with certainty; for the most part, appendicitis was thought of, and, on this account, laparotomy (with favorable result in a case reported by Watson-Cheyne).

The **diagnosis** of the condition presents no difficulties when there is blood vomitus or tarry stools, and one must also consider the possibility of ulcer of the stomach when gastralgia and symptoms of peritonitis are present; in an observation published by Frommer, the possibility of association of ulcer of the stomach with severe aphthous stomatitis was suggested, but, nevertheless, as already said, in most instances one can only conjecture.

There is seldom any definite indication for **treatment.** Exploratory laparotomy is indicated when the symptoms are severe, especially in cases of recognized peritonitis. When the clinical diagnosis of a case of ulcer of the stomach is a possibility, it should be treated at once by securing absolute rest for the stomach, external applications of cold and rectal feeding. Gelatin injections may be given, such as are serviceable in melæna. A 2 per cent. solution carefully sterilized (because of the danger of tetanus infection) should be injected, in quantities of 15–20 c.c. (3–5 dr.) into the subcutaneous tissue of the breast or thigh (in small children in two places), and the injection repeated in a few hours in case the bleeding does not stop. Careful diet and the use of alkalies (bismuth, alkali waters, etc.) are said to gradually assist the healing process.

Ulcers of the duodenum are even less frequent and their diagnosis is only conjectural. They play a certain rôle in the etiology of melæna neonatorum; but in later life they are most exceptional. I am able to refer to but one observation, that of Vanderpoel. This was a ten months old child which suddenly developed bloody stools and hæmatemesis, at the end of a chronic catarrhal colitis. The autopsy showed, as the cause, an ulcer the size of a bean lying close below the pylorus, in the posterior wall of the duodenum; the base of the ulcer was formed by the head of the pancreas grown fast to the duodenum.

2. FISSURE OF THE ANUS

This is an affection which is, apparently, especially common in artificially fed children suffering from constipation. It consists of a small loss of substance brought about through a mechanical injury of the mucous membrane of the anus. The ulcer is produced because of the perpetual stretching at every act of defecation, and the unavoidable infection arising in this region, partly through fecal masses and partly through the attempts to keep the part clean. The ulcer is situated, as a rule, behind the anal opening, in a line with the longitudinal folds of the rectal mucous membrane, for the most part on the posterior wall near the coccyx. In order to make it clearly visible, one must have the pelvis of the patient elevated, and the legs widely separated and flexed on the trunk, and must hold the gluteal folds apart; and, with the fingers placed flatly on both sides of the anus, press these apart, in doing which the outer sphincter is stretched and the mucosa pressed forward. This, then, appears slightly reddened and swollen, partially covered by a mucopurulent secretion, and, in the depth of the posterior rectal wall (seldom in other situations), is seen a more or less deeply fissured ulcer, which bleeds on touching and has a yellowish or grayish, coated base. Exposing of the ulcer produces great pain. At the same time, every mechanical irritation, as the act of

defecation, sets up a reflex spasm in the region of the sphincter, which already, in the attempt to open the anus, has most powerfully contracted.

The chronicity of this condition is brought about by a continuation of the constipation, which causes the production of hard and dry fecal masses. The removal of these masses from the rectum requires great exertion, and leads to a progressive deepening of the fissures, and eventually, also to the production of new fissures, and to a gradual hypertrophy of the external sphincter ani. The sphincter becomes thickened in these cases into a tense ring as hard as cartilage, behind which the deep fissure or several of them are situated as though cut out with a chisel. The slightest touch produces an intensely painful paroxysm which can be increased during the act of emptying the bowel to a convulsive seizure. When the defecation leads to such sharp paroxysms fecal retention is soon produced, which, naturally, still further increases the trouble. The micturition is likewise affected in many cases, either as retention or incontinence.

It is necessary for the **diagnosis** of the condition to make the fissure visible, in the manner above described. The presence of constipation, pain before, and during, or even a short time after, defecation, the appearance of blood, mostly in the form of streaks in the feces, suggests certainly a fissure although they can be produced by other causes. Moreover, there may be a loss of substance in the anal region, produced by eczema intertrigo. This, however, is situated, as a rule, on the outside of the rectum and not above the sphincter and does not cause such severe pain. In gonorrhœal disorders, especially vulvovaginitis, the process can be extended to the anus, and can lead to inflammation, with secondary ulceration of the rectal region. But this also is a process spreading from without, the association of which with the genital disorder is soon clear, and in any case can be verified by an examination of the rectal secretion for gonococci. Syphilitic processes are frequently localized in this region and ulcerations of the anal mucous membrane belong to the early manifestations of hereditary syphilis. The other wide-spread syphilitic lesions are a proof of the specific nature of the lesion. Broad condylomata, a late symptom of hereditary syphilis, and localization of acquired syphilis, frequently surround the anal region. They, however, are situated at a point of transition between the skin and the mucous membrane, are much more superficial, and are easy to diagnose because of the variety in their appearance, their color, and, as a rule, the presence at the same time of other syphilitic manifestations.

A careful examination of the rectum should be made with a well-oiled finger, protected by a condom, in order to find whether or not other causes, such as rectal polypi, foreign bodies, and the like account for the pain and the blood on defecation.

The **treatment** of these fissures is medical or operative. The indications for the medical treatment are causal and local; that is, it attempts to remove the causes of the fissure, and therefore the obstacles preventing its healing. The constipation should be relieved through proper nourishment, and a careful cleansing of the region of the anus should be insisted upon. This is best done, perhaps, with cotton tampons well anointed with vaseline, so as to diminish every mechanical injury from without. In addition, local manipulation should be considered, in order to modify the pain at every act of defecation, and by properly greasing the end of the intestine to effect the smooth passage of the stool. This is most satisfactorily accomplished through the use of suppositories containing some anodyne, which are introduced before emptying the bowel [for example, anæsthesin .25 Gm. (4 gr.), butyr cocoa q.s. to make two suppositories; or, cocaine muriat .03 Gm. (.45 gr.) to make 3 suppositories, extract of belladonna, in equal doses, in children of six months; double quantity later]. In the meantime, medication which produces superficial slough and so promotes cicatrization should be applied to the small ulcer. Felizet-Branca recommend for this purpose a daily enema with extract of ratanhiæ (1½ per cent. in water), or, 5 per cent. ratanhiæ ointment. I prefer to touch the part with silver nitrate, followed by the application of salt solution to lessen the pain of the treatment. By others, copper sulphate (5–10 per cent. solution), chloral (2–3 per cent. solution) are recommended, either as injection or irrigation. Treatment with orthoform, after previously carefully cleansing the fissure and pencilling it with a 5 per cent. solution of orthoform in collodion, has been recommended; still, because of the poisonous properties of this remedy, care should be exercised.

If all these methods are unavailing, and the fissure becomes constantly broken open and extends further into the tissues, if the sphincter becomes greatly hypertrophied, and the child, because of the pain and loss of sleep, suffers in nutrition and becomes anæmic and thin, then one is obliged to resort to surgical interference. Of the various methods recommended, namely, incision, excision and dilatation, the last is the least dangerous and most efficacious. It is carried out in deep narcosis and results, as a rule, after a few days in a complete cure. For a more detailed description of the method, and the after-treatment, the reader is referred to the text books on the Surgery of Children.

3. PROLAPSE OF THE RECTUM

Prolapse of the anus or rectum is present when, in defecation, the mucosa of the anus, a larger portion of the rectal mucosa, or even all the layers of the wall of the rectum, are extruded from the anus. This condition can lead to a constant protrusion of the part. It is

called a partial or complete prolapse according as to whether the mucosa alone or all the layers of the intestinal wall are involved in the prolapsus.

The latter condition may occur either when the lowest portion of the rectum is prolapsed, so that the skin of the anus passes without transitional folds into that of the rectum, or when only a higher segment of the rectum is prolapsed, and its lowest section, directly above the anus, remains in its normal situation so that a finger or sound introduced along the side of the rectum, after a short space, comes upon the reduplication of the mucous membrane, at which point prolapse begins.

The condition is seen more often in childhood, particularly in the second and third years. The following are the usual **causes**: diarrhœa with much tenesmus, especially in catarrhal colitis; constipation, which leads to a great increase of the abdominal pressure, hard fecal masses, the expulsion of which bulges the rectal mucous membrane forward; difficulty in micturition, which reacts upon the rectum; mechanical conditions of local nature, such as rectal polypi; inflammation of its mucosa; and, finally, severe paroxysms of coughing which lead to strong pressure, particularly whooping-cough.

To these causes should be added the anatomical disposition peculiar to childhood: namely, the narrowness of the infantile pelvis, its consistency, largely cartilaginous, the almost perpendicular course of the rectum, the greater play of its attachments with the neighboring organs, etc. Moreover, weak constitutions and chronic disorders of nutrition assist in the tendency, so that prolapse of the anus and rectum are present, for the most part, in anæmic, badly-nourished children.

I found an inflammatory thickening of the rectal mucosa and a cellular infiltration of the submucosa, in a case examined at autopsy, which produced a considerable loosening of the attachments between the single layers of the intestinal wall, and subjected the rectal mucosa to the action of the greatly hypertrophied muscularis mucosa. It seemed as if the contraction of this muscle-layer pushed the rectal mucosa forward, and that the latter, aided by the strong vis a tergo pulled the other layers of the wall after.

The **diagnosis** of the condition presents no unusual difficulties. It is easy to avoid mistaking it for a polypus or other tumor. Hæmorrhoids of such size are scarcely ever seen in children. An intussusception of the rectum, projecting out from the anus, can be diagnosed apart from the severe general symptoms accompanying it, by means of a digital examination, which shows that the rectum is in normal position, and the above-mentioned reduplication of the mucosa is absent, or cannot be reached.

The **symptomatology** is also fairly clear. At first, after every defecation a dark red cylinder protrudes, having on its surfaces trans-

versely arranged folds, and with a round lumen at the free extremity. Later on, the cylinder, which becomes larger, remains on the outside, and, when the prolapse involves all the layers of the intestinal wall, it has a tendency to bend slightly forward. The mucosa becomes redder, bleeds spontaneously on touching, secretes purulent mucus, and shows superficial ulcerations which are soon covered by yellowish deposits.

It is easy, at first, to reduce the prolapse, and to have it remain in place until again protruded by another act of defecation, an attack of coughing, or similar cause. In the later stages, however, it prolapses again immediately and it is no longer possible to hold it back. There may be so much increase in the obstruction, that the bowel can no longer be replaced when as a rule, a constriction takes place at the base of the tumor, at which point the mucosa shows more or less extensive necrosis.

This fact of a possible incarceration, which, however, seldom happens in children, together with the danger of hæmorrhage or infection, makes an otherwise good prognosis for this condition more serious.

The object of the **treatment** is to reduce the prolapse, and to hold it in place. The first procedure is much the easier and succeeds in most cases, except in those of incarcerated prolapse, a very rare occurrence in childhood.

It is best to proceed by covering the prolapsed portion of the rectum with a bit of gauze, well spread with vaseline, and by pressing slowly from the periphery, so to replace the tumor into the rectal cavity. This often permanently succeeds if one is careful that no constipation occurs, and that the child passes its movements with as little abdominal pressure as possible, as when it sits on the vessel with its legs hanging down, or while in a dorsal position. When this does not succeed, a bandage can be applied, as has been often recommended (by Basewi, L. Fürst and others).

Local treatment of the rectal mucosa, tending to contract it, can be tried. Thus, Hippocrates sprinkled a mixture of rind of pomegranate, cypress seeds and alum, upon the prolapsed mucosa. Lloyd strokes the part with lunar caustic, Woods, Allingham and others recommend touching it with fuming nitric acid, Broca, enemata of cold borax water. When these procedures do not succeed, the application of red hot iron, or thermo-cautery, must be considered, either linear or punctiform cicatrices of the mucosa are made at the point of transition between the anal epithelium and the mucous membrane. I have seen permanent cure result from both procedures. As has been said, an irreducible prolapse in childhood is exceptional, and, therefore, the operative removal of the mass, or resection of the prolapse, as has been performed by Heineke, Mikulicz, Hochenegg and others, is rarely considered. Broca, who has had large experience in the surgery of children has had, as yet, no opportunity to perform this operation.

It is evident that, after a complete and satisfactory replacement of the prolapse, care must be exercised to improve the child's nutrition, to overcome the anæmia, and to regulate the digestion.

4. CONGENITAL AND ACQUIRED DILATATIONS, CONSTRICTIONS AND OCCLUSIONS OF THE INTESTINE

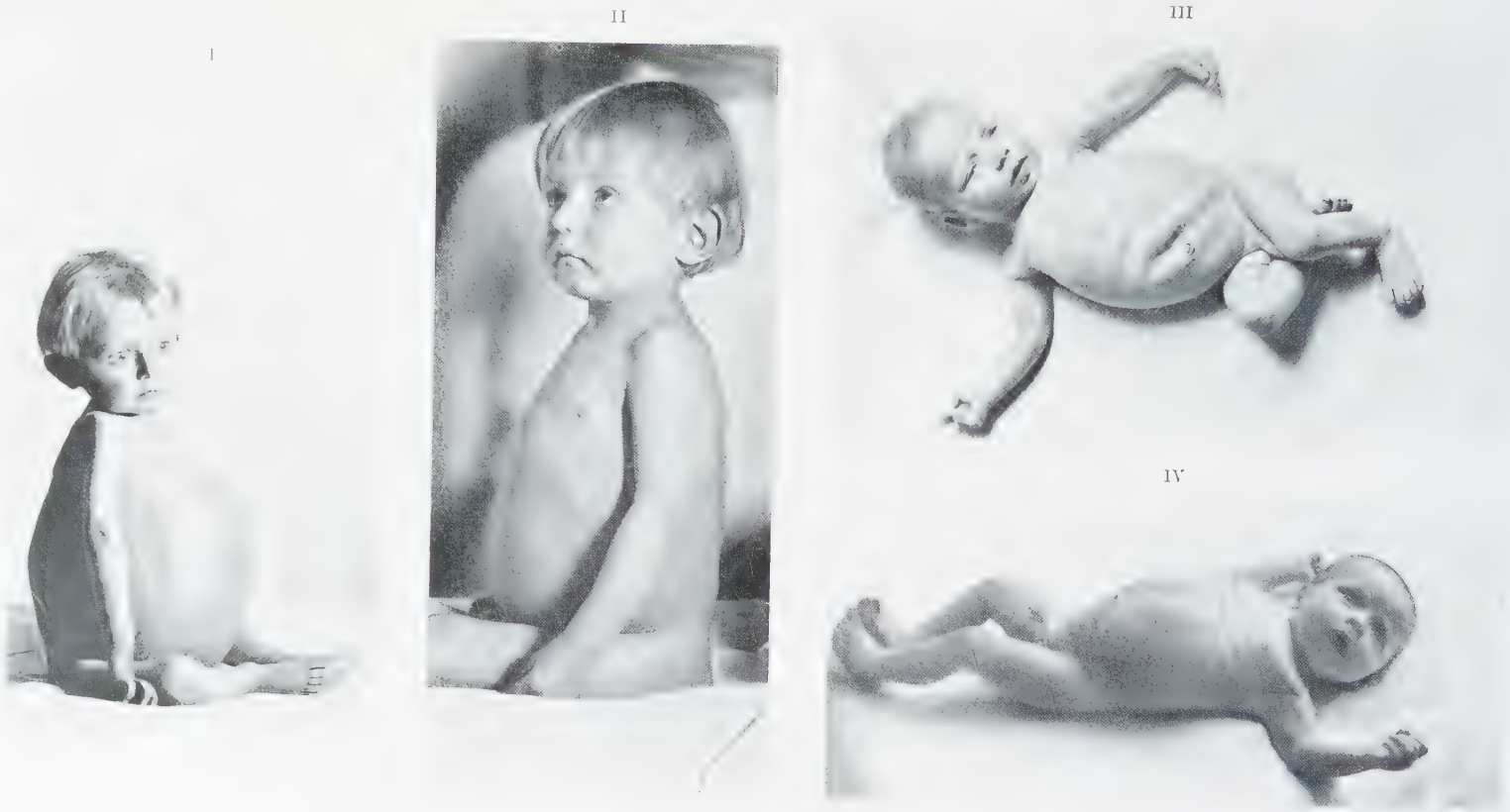
(*a*) HIRSCHSPRUNG'S DISEASE—CONGENITAL DILATATION OF THE COLON

This very interesting condition, the etiology of which is not yet entirely clear, was first described in 1880, by Hirschsprung, in Copenhagen. He states, in his last publication on the subject, that he has seen in all ten such cases. The literature with which I am familiar, particularly in the works of Concetti, Gourevitch, Neter, Ibrahim and others, is already rather considerable.

According to the **symptomatology** of the affection, one must distinguish *two groups of cases;* namely, those which show at birth, or in the first days of life, the phenomena of persistent constipation and gaseous dilatation of the abdomen; and, others in which these symptoms begin later, for the most part at the time of weaning, or at the beginning of accessory feeding.

The *first group* concerns children who are born apparently healthy, in which often after birth no external alteration is noticed, and to which the attention of the attendants is first aroused because of the fact that there is no spontaneous evacuation of the meconium. The physician, on being called in, convinces himself, by digital examination of the rectum, that there is no mechanical obstruction in this region. After giving an enema, a small quantity of infant feces is evacuated; the child remains restless, sleeps poorly, has no spontaneous defecation; and dilatation of the abdomen is added as a new symptom of the malady, the abdomen becomes tense, the skin is coursed by dilated veins, and is at the same time slightly œdematous. This condition increases visibly; the diaphragm becomes pushed upward, and dyspnœa and cyanosis may be present. If a rectal tube is introduced, and water permitted to flow in, the water comes back only in part, and the meconium is brought out in large quantities first through the introduction of the finger into the rectum and massage of the abdominal wall with the other hand.

The abdomen then sinks and becomes softer, and the condition of the child is improved; again it takes nourishment, the cyanosis disappears; but, after some time, the whole affair is repeated, and so it continues, until, finally, after a varied length of time, death intervenes, brought about, sometimes, by interference of the respiratory organs; sometimes, by insufficient nourishment or also by chronic intoxication, from decomposition products taken up in the blood-stream. In this last condition, coma, convulsions or other meningeal-like symptoms may result.



HIRSCHSPRUNG'S DISEASE (DILATATIO COLI CONGENITA).

I. Three-year-old boy before emptying of bowel. II. The same boy seated after emptying of bowel.
III and IV The disease in the first stage. (Peristaltic movements of the bowel.)

In contrast to these cases, which represent the true picture of the malady presented by Hirschsprung, there are other cases in which, after several months of complete health, or at most a slight tendency to costiveness, persistent constipation sets in, for the most part, as already mentioned, influenced by accessory nourishment or by weaning. The dilatation of the abdomen with gas soon follows, and continually increases, so as finally to present the appearance of a so-called balloon belly, which in size and form is not found in any other condition. (See photographs, Figs. 20 and 21, and Plate 43, Figs. 1 and 2). Moreover, the vitality of these patients is much endangered, and, in consequence of the marked constipation, ulcerative processes of the mucous membrane of the large intestine may develop, which lead to abscesses of the submucosa (Hirschsprung) and likewise can occasion severe cachexia.

Fig. 20.

Congenital dilatation of the colon. Before high irrigation.

The **etiology** of the affection is different in each of the two groups just briefly sketched. When the symptoms set in at once at birth, or shortly after it, we must assume as its cause a congenital malformation of the colon, which leads to considerable dilatation, eventually, also, to hypertrophy of its walls (which, however, is not constant), and so a stasis of the intestinal content with its sequelæ is brought about. These are the real cases of congenital dilated colon according to the conception of Mya.

The *second group* of cases begin first in the later months of life. In order to furnish a complete picture of Hirschsprung's disease in its clinical manifestation we must assume a congenital anatomical dis-

position of the sigmoid flexure (unusual length and tortuosity) which, indeed, means only an increase of the normal infantile condition; and which, under the influence of an alteration in diet, may lead to coprostasis, dilatation of the colon and hypertrophy of its walls; in short, to all those changes which we find already brought about in the first type.

The probability of such a method of origin, which has already been suggested by the anatomical findings of Marfan, Neter, Saias and others, is increased by an interesting clinical observation of Ibrahim, to whose kindness I am under obligations for the original notes of his case (see Figs. 3 and 4 on Plate 43). From this it is evident that the part of the intestine outlined on the intestinal wall, because of its great dilatation and peristaltic activity, corresponds to the widened and lengthened sigmoid flexure. This can also be verified by the introduction of an intestinal sound, and indicates with great probability the first stage of the disease.

Fig. 21.

Hirschsprung's disease (congenital dilatation of the colon).

The corresponding *anatomical findings* are somewhat varied; their reliability, however, suffers somewhat because of the fact that the cases come to autopsy often after the illness has lasted a long time. On opening the abdominal cavity, the colon is found dilated to the size of an arm and is bent up into two legs filling almost the whole peritoneal cavity. The length and free mobility of the mesentery of the sigmoid flexure can be ascertained. According to Hirschsprung, the walls of the very much dilated and often lengthened colon are always hypertrophied. (Figure 21 shows this plainly.)

Still this condition does not apply to all cases, so that Concetti distinguishes three types, which he designates as myerocolia (simple lengthening), ectocolia (ectasia of a more or less long section of the colon with or without compensatory dilation, or hypertrophy of the portion lying next to it), and, megalocolia (applying particularly to a general enlargement in the diameter of the lumen and to a thickening of the intestinal wall). In addition to clinical observation, anatomical experience, too, is in favor of a congenital origin of many cases. Such congenital dilatation or hypertrophy may also occur in other portions of the intestinal tract, as in the interesting case of a child who died in twenty-six days, reported by Schukowski, which, in addition to congenital dilatation and muscular hypertrophy of the colon, had a marked

increase in the musculature of the whole small intestine, and, at the same time, a stenosis of the duodenum and an umbilical hernia. There are moreover, also, cases of congenital origin which show only scattered areas of hypertrophy of the wall of the large intestine, which areas alternate with atrophic portions (Concetti); as well as those in which extensive thinning, and partial, or complete, disappearance of the muscle-coat in the walls of the colon is found (Beighing). I have already mentioned the frequent ulcerations of the mucous membrane and the submucosal abscesses (Hirschsprung) springing from them.

Even with the naked eye, it is possible to determine that the principal part of the thickening of the wall of the large intestine takes place in the muscular coat; and, in histological sections, one of which I am able to present from the preparation of Dr. Gourevitch (Fig. *n*, Plate 42), one can see that the hypertrophy and also the hyperplasia, as the measurement of a single cell indicates, concerns chiefly the circular muscle-layer and the muscularis mucosa. Among other microscopic changes are connective-tissue proliferation, especially in the neighborhood of the submucosa, dilatation of the vessels, obliterating arteritis, and leucocytic infiltration; and, also, a thickening of the serous coat, as made out from the researches of Concetti, Genersich and others.

FIG. 22.

Congenital dilatation of the colon. The preparation shows the great dilatation, particularly in its transverse diameter, of the large intestine and the hypertrophy of its walls.

The **treatment** of the affection has been attended, heretofore, with little success. Most of the patients, after the symptoms have lasted for a shorter or a longer time, die in consequence of weakness or chronic intoxication, from poisons from the sluggish intestinal contents. The first often very pressing indication for treatment consists in the evacuation of the fecal masses and the removal of the intestinal gas. To accomplish this latter result, Hirschsprung finally recommends a puncture of the intestine. Enemata and high irrigations, in which often a large part of the water introduced remains behind, either fail entirely, or are, at most, incompletely successful; and, therefore, must be accompanied by a manual emptying of the rectum and massage of the abdomen. In this manner incredibly large fecal masses are often emptied

out (in an observation of Concetti over 10 kilos; 25 lbs.) and the size of the lower abdomen is markedly reduced, as is indicated in the figures here reproduced (see Figs. 1 and 2 on Plate 43). When this method is not successful, one can attempt laxatives and glycerin suppositories (Fenwick, Levi).

This evacuation of the intestine must be frequently repeated and assisted by abdominal massage and electricity, in order to accomplish the least palliative result for any considerable length of time. Massage of the abdomen is best performed by beginning in the right iliac fossa and from there upward and across the abdomen, and then downward in the region of the left inguinal region; and should consist of a progressive kneading of the large intestine with the fingers and tapping it with the open hands, laid on in rapid alternation, and in thoroughly shaking the abdomen between the out-spread fingers, and similar procedures, each of which should last about five minutes, and which should be repeated every morning at about the same time.

To carry out the faradization, the electrodes are set side by side, and passed along the course of the large intestine; or, the so-called electrical lavage (Lenander) can be used, in which, first, the rectum is filled with salt solution and one electrode, covered with India rubber but with its point free, is introduced into the rectum and applied to the rectal mucous membrane, while the other electrode is moved up and down on the outside. In addition, a tight bandage is applied to the abdomen (Escherich). The administration of the tincture of nux vomica (Levi) and similar mechanical and medical methods, intended to increase tone can be undertaken together with a moderate diet, which should be as concentrated as possible. These measures may check the malady for a long time, and produce relatively satisfactory results. For a permanent cure of the abnormality, operative procedures have been recommended. The simple production of an artificial anus is rather purposeless, and, so far as I can discover, from the results in the literature, is shortly attended, as a rule, by a fatal termination. The much more rational resection of the intestine, in a case reported by Björkstén (an anastomosis of the colon and rectum was attempted) had a temporary effect, although the child died finally in the effort to close the artificial anus first made. Kredel operated more successfully, but still further experience must be relied on to indicate with certainty the proper procedure.

(*b*) INTESTINAL STENOSIS AND ATRESIA

There are congenital and acquired forms which will be described separately.

The **symptoms of congenital intestinal stenosis and intestinal occlusion** are violent vomiting, beginning shortly after birth, fol-

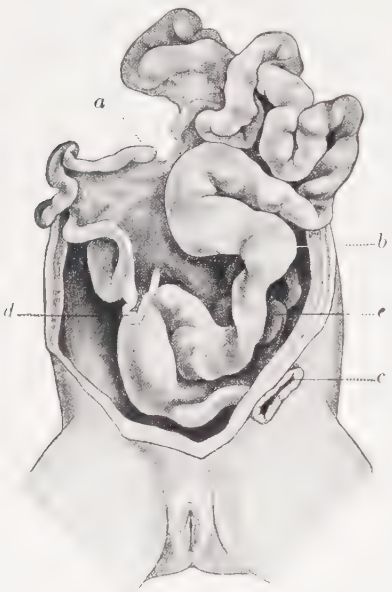
lowed by distention of the abdomen, and the complete failure to eliminate meconium per anum. The higher up the stenosis is situated the earlier the vomiting begins. The vomitus consists, at first, of the intestinal contents present, as far as the point of closure, then, also, of the nourishment given, and lastly of bloody material which comes from the stomach, the small vessels of which, in the mucosa, are ruptured in consequence of the great straining. When the stenosis is low down in the lower ileum, or in the large intestine, even fecal matter is passed by the mouth. The degree of distention depends upon the situation of the narrowing. The nearer it is to the stomach, the less is the dilatation, which, if the obstruction is close to the stomach, is limited to the epigastrium, while, in cases where the interference to the passage of the stool lies further down, the abdomen is greatly distended, particularly in the region of the navel and shows a slight flattening in the flanks (Nobécourt). To these symptoms, disturbances of micturition are often added; this, either because of the complete stoppage of the resorption of fluids, stops entirely, or becomes irregular and painful, which is occasioned by compression of the ureter by the greatly distended intestine, or must be regarded as a reflex symptom. The great gaseous distention of the abdomen elevates the diaphragm which again may lead to dyspnœa and cyanosis.

The appearance of the child indicates the gravity of the affection. The face becomes sharp, the expression is drawn, and distorted by pain, the tongue dry and the skin cool [temperatures about 35° C. (95° F.) are very frequent]. Wasting is rapid, and, in this state of collapse, interrupted by tonic spasms or convulsive seizures, death intervenes in a few days (generally in 4–5, seldom a week, exceptionally longer). As a rule, on examination of the abdomen, no resistance is present, and no tumor is felt. Violent peristaltic movements are visible, under the tense and attenuated abdominal wall, particularly when the stenosis is placed low down. In such cases gurgling and rumbling is heard and felt. The examination of the rectum, which should never be omitted, in order not to overlook any occlusion in the neighborhood of the anus or rectum, shows its complete patency, even in the case of a stenosis situated low down. There may be some widening of the ampulla; the palpating finger, however, or the sound rarely comes upon an obstruction. (In single rare instances the obstruction is occasioned by kinked sigmoid flexure, which is pressed deep in the pelvis and bulges forward over the rectum—Kuligo, Mercadi).

The **etiology** of congenital intestinal stenosis and occlusion is not always the same. Kreuter has, it is true, made the attempt to explain all the various forms by the history of their development, supported by the fact that the intestinal canal in most vertebrates and in man possesses in early embryonic life a well-developed lumen, which later

is covered by epithelium, in order that it may again finally become patent. He assumes that in such cases the opening does not take place at all, or only in an incomplete manner when atresia or stenosis takes place. He considers these conditions, therefore, as simple phenomena of retardation, and thinks that the factors emphasized by others as important etiologically, such as inflammation, intussusception, volvulus and the like, are secondary results of the primary processes. This appears to me to be a little too schematic, especially as in this way many other findings, such as anomalies of the vessels, obliteration of the blood-channels of the intestine, etc., are not explained.

Fig. 23.



Congenital atresia of the ileum. *a*.—The two blind ends of the ileum. *b*.—The greatly dilated jejunum. *c*.—Opening of jejunostomy. *d*.—Vermiform appendix.

It is a fact that traces of fœtal peritonitis, of possibly syphilitic or tuberculous origin, are not infrequently found; and, moreover, the persistence of the ductus omphalomesentericus can lead to strangulation of the intestine and to occlusion of its lumen. To account for many cases, ulcerative inflammation of the mucosa, with subsequent scar formation, must be assumed as the cause. Other cases are explained by intussusceptions, which arose and were reduced during intra-uterine life (Chiari, Braun), and, still other causes, by compression by congenital tumors of the abdomen (Schukowski), anomalies of the circulation with ischæmia of certain parts of the intestine, or, by obliterating arteritis resulting from inflammatory infectious processes of placental origin (Cordes). Amniotic bands may be present, by which occlusion occurs in the form of a transverse septum, to explain which one must assume a productive inflammation of the intestinal wall. Finally, there are instances in which the most careful macroscopic and microscopic investigation fails to discover any cause.

According to the **pathological anatomy** of the condition, stenosis and atresia of the small intestine stands first in frequency, next are those which are localized in the duodenum, and last come those of the large intestine. The intestine appears much reduced in lumen, or this is absent completely for a section, and replaced by fibrous tissue; or, finally, both ends of the portion of the intestine concerned terminate in blind sacs, which are held together by mesentery. The section of the intestine above the stenosis appears greatly dilated, the portion below

it completely collapsed. (All these types are clearly seen in the illustrations, taken from the preparations in the collection of the Pathological Museum, and, for the privilege of reproducing them, I would especially thank the Director of the Institute, Prof. Chiari—see Figs. 3 and 4 on Plate 44.)

In most cases, no malformations are found in the rest of the body but not infrequently, in the region of the intestine, such abnormalities as anomalies in position, an unusual shortening of the intestine, the absence of certain parts, and similar alterations can be demonstrated. Moreover, multiple stenoses have been repeatedly described. (The exact details of more statistical interest are to be found in the collected publications of L. Cordes, Weill, Pehu, Bossowski, and others.) I would call attention to an interesting finding that was discovered on examination of the intestinal content: this was found to be sterile below the point of atresia (Bossowski, Wyss, and others), while the contents above it contained B. coli and other varieties. This renders probable the assumption that, in the physiological process, the bacterial infection of the intestine takes place by the mouth and not by the anus.

The **diagnosis** of this condition, in so far as it rests upon the recognition of a stoppage of the bowel movements, is relatively easy. The vomiting, beginning soon after birth, the meteorism, in addition, and the failure to evacuate meconium, are such striking symptoms that no serious doubt can arise.

In stenosis of the œsophagus, or closure of that passage, which can be determined by probing, the nourishment is returned immediately after drinking. In pyloric stenosis the vomitus is never colored with bile, or fecal in character, and the symptoms are developed more slowly; frequently, the movable tumor, corresponding to the hypertrophied pylorus, is palpable. It is true that it is rarely possible, clinically, to certainly distinguish a duodenal stenosis, near the stomach and above the ampulla of Vater, from a pyloric narrowing. I have already mentioned that by careful examination of the anal region, and of the rectum, one must exclude any obstruction in these parts. Finally, fœtal peritonitis must be mentioned, as it also leads to vomiting and meteorism, but in this condition there is no absolute constipation; on the contrary, there are diarrhœa stools; the movement of the fluid in the abdominal cavity is soon evident, and the sensitiveness, on palpation, as well as the fever, which is rarely absent, all point to the inflammatory character of the disease and to its localization. The exact situation of the stenosis can be conjectured, from the factors mentioned in the description of the symptomatology, but it can not be certainly determined.

The **prognosis** of congenital intestinal stenosis and atresia can be designated as absolutely fatal. I can find no single instance in which

there has been a successful operation performed, and certainly nothing is to be expected from internal medication.

The **treatment** can only be operative, and should be undertaken as early as possible. In view of its absolute failure, it is questionable whether it would not be better to spare the little sufferers the useless annoyance. Tuffier who (according to Nobécourt) collected 32 operations (26 of which were enterostomies, 4 entero-anastomoses and 2 perineal ileostomies), could not bring forward a single successful case. Braun, who reported 25 operations, had the same results, and Bossowski, likewise, in two cases of his own and 31 taken from the literature. I have myself, in addition, gathered together a number of observations, not elsewhere reported, in which various surgical procedures were undertaken (such as the production of an artificial anus, suture of the large intestine with the rectum after resection of the portion in atresia, entero-anastomosis, etc.), and they all, also, ran a fatal course.

There are, however, cases of congenital stenosis of a less complete type which produce no disturbances which can be recognized clinically, during the first months of life, and first, later, under the influence of a change in nourishment, or other factors, show the symptom-complex of occlusion. Thus Lees and Thursfield had a child of eighteen months under observation, which was previously in a perfectly normal condition, but which then had attacks of pain and diarrhœa, with bloody stools and vomiting, followed by complete obstruction with meteorism and violent vomiting. It was discovered, on section, that the cause was an apparently congenital tuberculosis with numerous points of stenosis of the intestine and adhesions between the abdominal organs. Hey and Grewes saw a stricture, three inches long, of the small intestine, which hardly permitted the passage of a probe, in a boy of twenty months. Similar cases have been several times reported but they can not be said to have a symptomatology, and a diagnosis can hardly be made during life.

The **acquired intestinal occlusion**, apart from intestinal intussusception, which, because of their characteristic symptoms, will be separately described, are produced by volvuli of the intestines, by the constriction of single intestinal loops by means of Meckel's diverticulum, by the incarceration of the intestine in congenital open spaces in the mesentery, or in retroperitoneal recesses, by strangulated hernias, not visible from without (specially crural hernias), and like conditions. (Such cases have been reported by Frölich, Schochner, Snow, Bell, Burgess, Janero and others.)

As a rule, the development of symptoms of intestinal occlusion begins suddenly, more seldom after prodromal manifestations, in the form of diarrhœa with bloody discharges, colicky pains, or after mechanical injury of the abdomen, through convulsions, a blow, or the

like. It may not be possible, on examination, to determine the nature of the preceding disturbance. A long mesentery provides the anatomical predisposition to volvulus, which occurs, however, comparatively seldom in childhood. Rarely a tumor is the cause (as in the case of a cyst of the mesentery, reported by Blum); the other factors are clear in their anatomical bearing, from what has already been said, without further explanation.

The **prognosis** of such cases is, in general, considerably more favorable than in the congenital stenosis, as an operation undertaken promptly has resulted in cure in the majority of instances. The simpler are the conditions present, just so much more favorable are the chances; on this account, they are particularly good in cases of constriction by Meckel's diverticulum, the severing of which quickly and permanently removes the obstruction; while, strangulation in a peritoneal recess, or volvulus, because of the great injury of the intestine already taken place, makes the operation more difficult and renders the outlook less favorable.

(*c*) INTESTINAL INTUSSUSCEPTION—INVAGINATION

As a rule, this is brought about through the invagination of a part of the intestine into another part situated below it (so-called descending type), more seldom the invagination takes place in the opposite direction (ascending type).

A tumor produced in this manner consists of a central canal and three lateral cylinders, lying parallel around the canal (Jalaguier); the outermost cylinder, the intussuscipiens, possesses an external serous and an internal mucous surface, which latter is turned towards the mucous membrane of the middle cylinder. The middle cylinder represents a fold of the intestine, and is continuous at the border of this fold with the inner cylinder. The point where the outer cylinder turns into the middle cylinder is called the neck of the invagination; and where the middle turns into the inner cylinder is called the head of the invagination. The mesentery is soon drawn into the tumor, and, situated between the middle and the inner cylinders, exerts traction upon the intussusception, which is thus both limited in the direction of its long axis, and is curved with its convexity towards the attachment of the mesentery, so that the lumen, in this manner, is flattened and shoved to one side.

In addition to this simple (three-fold) invagination there is a double one, which arises through the folding-in of the first intussusception in the neighboring part of the intestinal canal so that the tumor then posseses five thicknesses of intestinal wall; a triple invagination would possess seven layers, but these last are rather rare.

The *mechanism* of the invagination is as follows: an intestinal fold, either through its own weight, or because of its contents, or from

the pressure or the pull of a foreign body lying in it, sinks itself in the neighboring portion. Anomalies of peristalsis may also play a part. These are the only factors in the ascending form, and, through contraction and stiffening of a portion of the intestine, enable this segment, following the course of the peristaltic waves, to bury itself in a neighboring portion at rest, or relaxed. Of course, these factors often work together, and, in a particular case, it is not always easy to determine the ultimate causes of the presence of the intussusception.

Apart from the varieties arising during life, and which alone interest us here, the invaginations formed at the death-agony are to be distinguished. These arise at the last hours of life, under the influence of irregularities of peristalsis, and show, on post-mortem investigation, no reaction of inflammatory nature.

According to the portions of the intestine invaginated in each other, one distinguishes ileocecal, colic, ileocolic and iliac intussusceptions, the nature of which is indicated from the names without further description. These various forms differ in frequency, thus, for example, in children the ileocecal type is most common. (It includes according to Leichtenstern 44 per cent., Clubbe 70 per cent., Grisel 82 per cent., and to Wiggin 89 per cent. of the cases.) Grisel, who has made thorough studies of invagination in childhood, gives the frequency of other types of intussusception as follows: ileocecal 11 per cent., small intestine 5 per cent. and large intestine 2 per cent.

As has already been mentioned, the traction which the mesentery, included in the intussusception, exerts, limits its extent in its long axis, and further produces, at the neck of the invagination, signs of strangulation and inflammation of the parts of the intestine involved; and, in consequence of this, partial adhesions are produced, and thus, likewise, further progress is prevented. The more acutely the invagination is formed the shorter it is. The length of the invagination depends, also, upon the portions of the intestine concerned. Thus, for example, the ileocecal intussusceptions are by far the longest; the head of these invaginations may extend into the rectum or be prolapsed from the anus, and there are some of this kind which have invaginated the whole of the large intestine. Next to these are the intussusceptions of the colon, while those of the small intestine are the smallest, but these, however, produce more often the double or triple intussusceptions.

The great frequency of invagination during childhood bespeaks a particular and especial disposition at this age. The greatest number of cases occur during the first six years of life. All statistics indicate that boys are more often concerned (about three times as frequently as girls). The slight attachment of the cecum in the iliac fossa, the active peristalsis, the weak development of the musculature and of the elastic tissue of the intestinal wall, render the invagination of single parts of

the intestine into neighboring folds easier in childhood than in later life. Moreover, the influence of race seems to play a part, since, in contradistinction to the large number of contributions which come from England and America, the reports from Germany, France and Austria are much less frequent, notwithstanding a greater tendency to write in these countries. Riddell has recently pointed to the occurrence of invagination in families; he observed the affection in three brothers and sisters.

To these predisposing causes, catarrh of the intestinal mucosa, such as can be produced in consequence of improper nourishment, is added as an immediate cause; likewise, constipation furthers the development of invagination. Among mechanical injuries must be mentioned traumata, particularly those which strike the abdomen directly, enemata given with improper instruments, an impressive example of which I have published; tumors of the intestinal mucosa, particularly polypi, or chronic affections of the mucosa and submucosa lead to overgrowth (Lawrence). A frequent mechanical cause of the origin of intussusception occurs when Meckel's diverticulum inverts one loop in upon another, particularly when a tumor is situated at its extremity, which not infrequently occurs; or, when particles of food are found in its lumen and which it attempts to remove by energetic contractions.

The vermiform appendix acts, also, in an analogous way. Ackerman, who has reported 12 observations of this kind, considers the inversion of the appendix the primary cause, and agrees with the generally accepted views that the production of this condition is favored by peristaltic stiffening, due to abnormal intestinal contents, adhesions with the neighboring organs, chronic inflammatory processes of the tortuous wall, and similar conditions.

According to the origin and cause of intussusception, one distinguishes acute, subacute and chronic forms, of which the last is only observed in later childhood, while the acute and subacute types occur particularly during infancy.

On **anatomical examination** of intussusception, one finds generally, above the tumor, a dilated portion of intestine, and, below the mass, contracted bowel (see Fig. 24 and Fig. *p* on Plate 42). If it begins acutely, symptoms of incarceration appears for the marked contraction in the region of the neck of the intussusception and the traction of the mesentery lead to œdematous infiltration of the intestinal walls with stasis and bloody exudation. The longer the strangulation lasts the more severe becomes the inflammatory swelling, to which is added pressure necrosis and gangrene, which takes place for the most part in the inner cylinder, particularly about the head and neck of the intussusception. Moreover, adhesions between single serous and mucous surfaces are formed, and finally, by the continuation of the strangulation, the delicate tissue is torn in one or

more places, so that one can see, as through a sieve, the intussusception, which, otherwise, is not visible from without. It is evident under these conditions that the neighboring portions of the intestine, especially the part lying above the obstruction, and the peritoneum will be involved in the spread of inflammation. The gangrene of both inner cylinders can completely destroy them, and in this way a kind of spontaneous healing may take place, since the involved portion can be discharged per vias naturales as a decomposed, dark, foul-smelling loop of tissue. In the meantime, sufficiently firm adhesions between the outer cylinders and the neighboring parts may be formed, so that the lumen is again established, although, since in any case, a deeply ulcerated mucosa is present at the seat of the previous invagination, cicatricial stenoses are rarely absent. Intestinal intussusceptions extending deeply into the rectum, and such as appear partially at the anus, lead to more extensive dilatation of the rectum and complete relaxation of the sphincters. The longer the invagination exists, the less pronounced become the symptoms of reaction in its neighborhood. These, in chronic intussusception of the small intestine, can be limited to moderate congestion. On still longer duration, and the intervention of symptoms of incarceration, all the changes described above may develop, and the intussusception becomes irreducible. Jalaguier compares the process alluded to with chronic and, at first, easily reduced hernias, which become suddenly incarcerated.

Fig. 24.

Invagination of the cecum. *a*.—Necrosis of the intussuscepted portion.

Ray has observed primary rupture of the intestinal wall in a case of intussusception arising acutely, as a result of the strong pressure with which the intussusception was brought about.

The proper recognition of the condition is of great importance since the chance of cure becomes worse the longer the duration of the intussusception. The symptoms are for the most part so clear-cut that the diagnosis offers no unusual difficulties. One must here separate the acute from the chronic forms because these differ markedly from each other in their clinical manifestations.

The *acute invagination,* as its name implies, begins suddenly in the midst of perfect health, indeed there are observations where children became ill during sleep; or, moderate diarrhœa, painful defecation and similar symptoms follow an injury. It begins for the most part with pain, which in younger children leads to restlessness and loud out-cries. The legs are drawn up against the abdomen and nourishment is refused. Older children, who can localize their pain, speak often of a definite place, which generally corresponds to the point of invagination. (A boy, about two and one-half years of age, once said to me that a flea was biting him on the right side of the abdomen.) One often observes, moreover, a point of particular sensitiveness from which pain radiates in different directions (toward the navel, the bladder, the genitalia, etc). The pains and the reaction phenomena vary according to the situation of the invagination. In intussusception of the large intestine they are very severe, uninterrupted, and followed by the quick collapse of the patient, since in this form the symptoms of incarceration set in quickly and increase. In other types they have a more paroxysmal character which is apparently associated with strong peristaltic movements. During the attacks of pain the abdominal muscles are tense and cramped, but after the pain has subsided the abdomen again becomes soft and is scarcely sensitive to pressure.

Vomiting is also frequent, but as Heubner has lately again pointed out, it is by no means a constant symptom. It begins relatively early. The vomitus at first consists of food, later it may become mucoid and bilious, and finally, because of the unusual strain upon the stomach mucosa, the vomitus may be mixed with blood. It has rarely a fecal character; often it continues from the beginning to the end of the illness, again, it may usher in the attack and afterwards cease, or it may occur only now and then during its course. There are cases described, however, in which vomiting, at first, unimportant, later could not be checked. It is the intussusception of the small intestine which is accompanied by marked stomach symptoms.

One of the most important signs is the appearance of *blood-stained mucus,* or stools consisting of pure blood. The blood springs from the invaginated portion of the intestine, and can be evacuated in such quantities as to be fatal. As a rule, however, this is not the case, and this symptom is limited to the passage of from one or more bloody, mucous stools, which finally, especially in the low situation or in the descent of the invagination, can be accompanied by tenesmus, so that the condition is similar to dysentery. This melæna is so frequent in intestinal invaginations during childhood (indeed Jalaguier says it never fails), that when it is present it possesses a pathognomonic importance; the failure of it, however, does not exclude the invagination. My experience agrees with that of Vernon and Audèoud, who saw a number of these

cases without this symptom. Often the bloody stools appear for the first time so late in the illness that they are of no diagnostic value.

In addition to these blood-stained, mucus masses, a certain amount of fecal material is also evacuated, and, from time to time, gas is passed. More seldom there is remarkable absolute obstruction of the intestine, such as is seen in the other forms of intestinal occlusion. This is present, particularly in the invagination of the small intestine. Where the process goes on to gangrenous decomposition of the intussuscepted bowel, and extrusion of the same per anum, normal passages may be resumed after the expulsion of the dark, foul-smelling shreds of tissue which often still retain the form of the bowel.

Invagination exerts its influence also upon micturition, as does every localized painful change in the region of the intestine. This may be expressed by dysuria and pronounced diminution in the amount of urine. This occurs in all types and therefore possesses no significance of local diagnostic value.

The *general condition* of the child is soon altered, in a manner very characteristic to one of even moderate experience; there is a peculiar prostration which is out of proportion to the severity of the symptoms at onset; the features become sharp, the expression of the face anxious, and soon the true facies abdominalis is noticed, which arouses the attention of the physician. In addition, cyanosis and dyspnœa are often present; the pulse becomes small and frequent and the temperature subnormal. Tonic contractures of the extremities occur and in infants, convulsive seizures may complicate the picture, until, finally, collapse or pneumonia ends the scene, or it may be peritonitis, with an increase in temperature, carries the child away.

The *examination* of the abdomen should be made during an interval free from pain, for in an attack such an examination is not possible because of the strong tension of the abdominal muscles and the great sensitiveness. A particular point of pain can be ascertained localized in a definite situation, and an oblong, sausage-formed, slight curved mass lying under the abdominal walls can often be felt in this region. The form of this mass is sometimes altered during manipulation, and on firm palpation it may produce a gurgling sound. When permanent contraction of the abdominal wall and great sensitiveness make an exact examination impossible, narcosis can be employed, by the help of which the diagnosis of the tumor can usually be successfully made. At the same time however, there are a number of cases in which such a mass cannot be felt, after combined examination from without and per rectum, or after anæsthesia, and only the whole picture of the illness, and the manner of its development could suggest the diagnosis of an invagination.

The facts become much more simple and certain when the intussusception is situated low down, so that the palpating finger can reach

it from the rectum. It feels like a polypus, or the soft vaginal portion of the uterus, but neither a peduncle nor the transitional folds can be made out as in prolapsus. In an invagination, situated sufficiently low, the finger comes upon the slit-like lumen, placed, for the most part, at the side, or feels two openings (in the case of ileocecal invagination with inversion of the appendix). On removing the finger, one finds it covered with bloody mucus, which trickles out of the relaxed anus, and which under the microscope is seen to contain red corpuscles, leucocytes, and numerous intestinal epithelial cells.

Not infrequently the invagination comes still lower, so that it finally extends from the anal orifice as a dark red, slightly bloody mass, over an inch in length, on the surface of which are ulcers covered with a greenish gray deposit. In appearance the mass resembles a prolapse of the rectum. It is, however, distinguished from the latter by absence of the reduplications, and by the severe symptoms of intestinal occlusion which mark its onset. Other signs of intestinal invagination, as a rule, precede the descent of the intussusception by several days (more seldom three to four generally five to six).

The phenomena of *chronic invagination* are much less striking. Often they are not recognized, and then only in the later stages when the symptoms of incarceration have already set in. There are indeed attacks of pain in the abdomen, still these are not severe and are usually separated by long intervals; vomiting is rather an inconstant symptom; the stools are at times constipated, at times loose; in the latter instances they are mingled with mucus or accompanied by tenesmus, so that one thinks of catarrh of the large intestine. Constitutional symptoms are few: the abdomen is soft, not sensitive; a tumor is not often felt. If one is palpated the resistance is very little increased. Its situation changes during the examination and it disappears when the intestine lies deeply in the abdomen. It is, of course, quite different when a tumor is palpable per rectum, or prolapses at the anus, the recognition of which removes all doubt. The prolapsed chronic invagination produces much less congestion than the acute, and, what is particularly important, is easily replaced. The first symptoms of oncoming incarceration increase the congestion, and produce a secretion of mucopurulent masses and hinder its reduction.

The course of an acute intussusception can be intense throughout; a fatal result may result in a half a day after the beginning of the symptoms. Generally, however, it takes almost a week before the symptoms have reached this height, from severe intoxication by poisons absorbed from the intestinal tract, or as the result of complications, or from peritonitis. In older children the disease may be prolonged into the second week. Spontaneous invagination, before the occurrence of the more severe symptoms of incarceration, or the formation of firm

adhesions between the single layers of the intestine, can occur (Attlas), but is a very exceptional termination. Stenosis of the intestine, after spontaneous sloughing of the gangrenous intussuscepted portion is also described (Orange and Hau). In any case, the relatively favorable terminations referred to, which may, however, be altered through peritonitis, pneumonia, septic infection and similar causes, coming on later, are not to be counted on. Acute intussusception, left to itself, can be regarded with certainty as having a fatal result.

The **prognosis** in chronic invagination is somewhat more favorable. This form may last for months or years, but here, too, a spontaneous cure can not be expected; and, during every portion of its varying course, there exists the danger of incarceration and the imminent risk to life produced by it. The importance of the recognition of this form is evident when the relatively favorable outlook, following timely operation, is considered.

From the reasons above mentioned the necessity of an **early diagnosis** is urgent. This diagnosis is made with great probability when a child, previously well or suffering from mild digestive disturbances, is suddenly taken ill with violent paroxysmal pain in the abdomen, passes no stool, vomits frequently, and discharges blood or bloody mucus masses by the anus. A tumor of characteristic form and position, palpable in the abdomen, renders this assumption still more plausible. A tumor palpable by rectum, or visible at the anus, makes the diagnosis certain.

The **differentiation** from intestinal occlusion brought about by other factors, such as volvulus of the intestine, constriction of the bowel by adhesions formed in peritonitis, persistent ductus omphalomesentericus and such causes are not always possible. The factors in differential diagnosis given by individual authors (for example by Jalaguier), such as the absence of a tumor and bloody evacuations, as well as the absolute occlusion by interference with the feces, arising through some other cause than intestinal intussusception, do not always hold as I have previously pointed out, since they all can occur in invagination, for example, in intussusception of the small intestine. Moreover, the confusion of these forms of occlusion is not of so much importance, since in every case early operation is advised.

In acute inflammation of the vermiform appendix, bloody stools are not seen, and the tumor does not appear to be so deeply situated as in intussusception, but more in contact with the skin lying above it. From this point it may extend into the depth of the pelvis and is, as a rule, recognized by manual examination from the abdominal wall and from the rectum as a diffuse, tumor mass, not cylindrical in outline. However, as many mistakes are unavoidable, one must bear appendicitis in mind because of its much greater frequency.

Much more serious consequences can result if the condition is confused with dysentery, and purgatives are given in order to empty the colon and to lessen the tenesmus. It is self-evident that this procedure leads to an increase of the symptoms of incarceration, and adds materially to the danger. I believe that in the examination of the bloody mucus, passed by rectum, which often contains entire bands of unchanged sloughed, intestinal epithelium, we possess a pretty good means of excluding dysentery, in which the stools are full of leucocytes and bacteria, and poor in epithelial cells. Moreover, the course of the two processes is rather different; particularly the initial fever, which is scarcely ever lacking in dysentery, is not present in intussusception. The severe general symptoms come on much sooner in dysentery and stand in no relation to the intestinal symptoms; the pain is not so localized, a tumor is never felt.

Confusion of invagination extending into the rectum, with a tumor or a polypus, is easy to avoid since the consistency, absence of a peduncle, the demonstration of a peripheral lumen, and the secretion of bloody mucus indicate that one has to do with prolapsed intestine. Likewise, a rectal prolapse is readily recognized as such, since it can be replaced, and extends either directly into the anal mucosa, or, if not, the transitional fold is felt a short distance above the anal orifice.

It is much more difficult, as I have already intimated, to make the diagnosis of chronic intussusception, and it is frequent to mistake this form for chronic enteritis, appendicitis, and the like. In every case a careful examination, which is directed particularly to the finding of a characteristic movable tumor, one which contracts and gurgles on palpation, and the rectal indications, also, should never be omitted. Broca, Moizard, and Gaudeau have pointed out a very important symptom in my estimation, the gaping of the anus, one already emphasized by earlier writers. This is never found in inflammation of the sigmoid flexure, nor in chronic intestinal catarrh, nor in the course of a low grade of peritonitis. This symptom led these authors to the proper recognition of two cases, and to their cure by operation.

Increase in pain, more violent recurrences of the attacks of colic, profuse evacuation of blood and mucus, violent vomiting and severe general symptoms point to incarceration. Increase in meteorism, as well as a rise of temperature, are signs of beginning peritonitis, further indications of which soon set in.

The **prognosis** of acute invagination is practically hopeless, unless therapeutic measures are undertaken promptly, for the possibility of a spontaneous freeing of the bowel, or recovery by gangrenous demarcation, as already described, are hardly to be considered. The most favorable therapeutic results are obtained according to Frisch in ileocolic invagination, the mortality of which only reaches 32 per cent.,

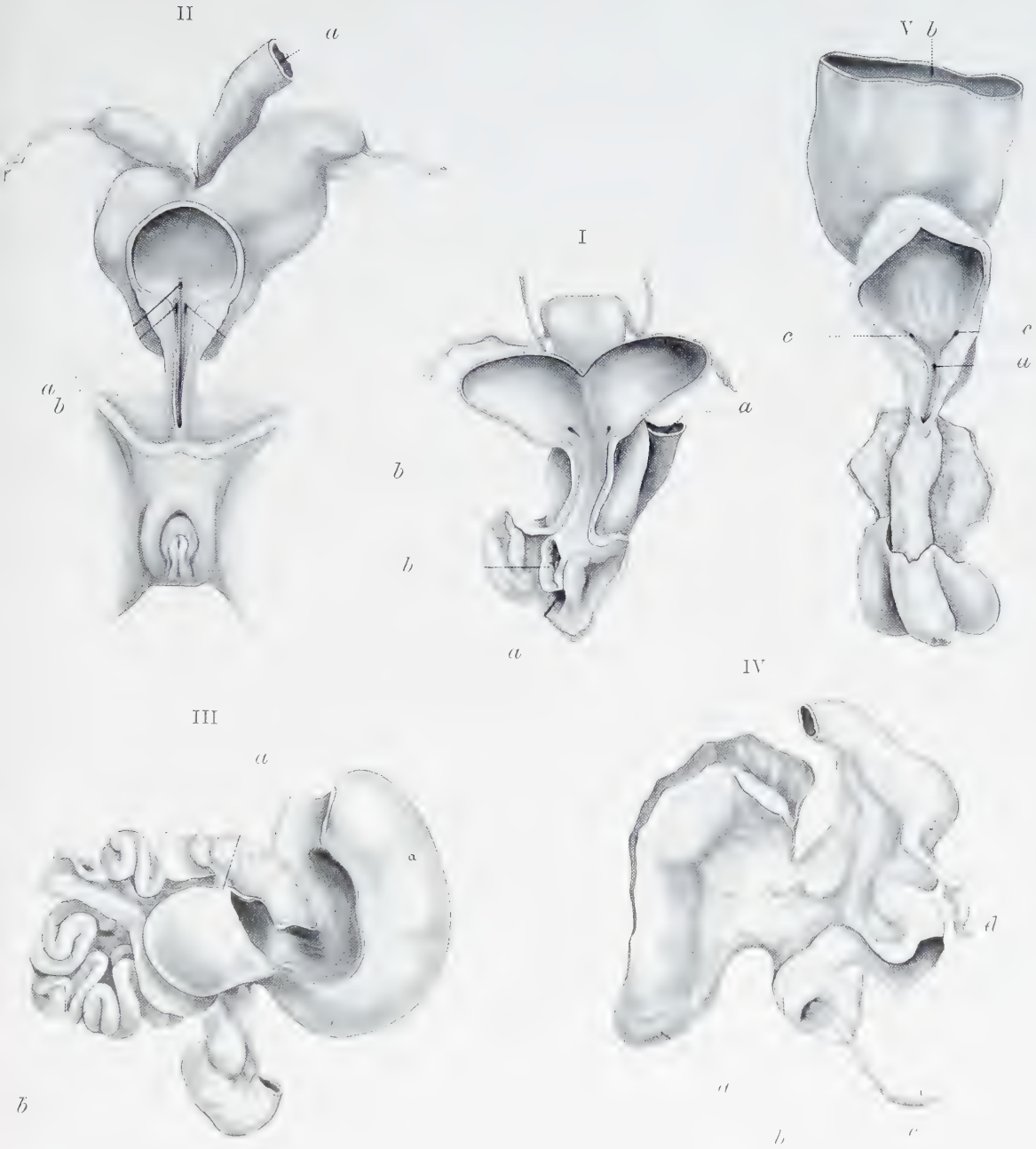
while it increases to 39.5 per cent. in the ileocecal type, and to 50 per cent. in cases involving only the small intestine. Chronic invaginations, in the cases collected by him, had a mortality of but 19 per cent. Heaton, in 104 cases collected, which were operated upon early, found that 66 ended fatally. Particularly unfavorable were the results in young infants, especially in those in whom reduction was not possible after the laparotomy rendering resection necessary. (Only 2 cases recovered out of 24 instances of this character).

Treatment has only one object, to reduce the intussusception by medical or by operative methods. In recent years under the influence of increased confidence in aseptic techinque, the tendency has been not to tarry with *manipulations of a bloodless* nature such as irrigations of water and of air and oxygen insufflations, but immediately to open the abdomen. I hold that this procedure has gone too far, judging from my own experience and that of many others (Clubbe, Wilkinson, Hond, Eve, etc.), and would recommend in fresh cases, which have lasted only a few hours from the beginning of symptoms to the time of observation, the careful use of an enema [a litre (one quart) of lukewarm water allowed to flow in from a height of $\frac{3}{4}$ metre (2 feet) while the pelvis is elevated]. Such a child must remain under observation after the reduction has been brought about, since the intussusception may form very shortly again. In addition, intestinal rest, secured by opium and continued as long as possible by means of liquid nourishment, is strongly indicated. The ballooning of the intestine, with air or oxygen, I consider as less worthy of recommendation; occasionally, repeated rupture of the intestine has occurred during this procedure (Godlee).

Consequently, I agree with most authors, that one should not persist in these manipulations referred to, and, when the first attempt is not successful, should resort at once to operation, for which preparation should be made in advance, and, on this account, the transfer of such patients to a hospital is recommended. The release of the invagination, during narcosis, is an exceptional but a gratifying occurrence, which I have once experienced (with permanent result), and which has also been reported by others.

The *dilatation of the intestine*, by injection with water or air, is used by many writers as a procedure preparatory to operation, intended to render the release of the intussusception easier. Whatever the choice of method may be, the simplest and shortest procedures are best since every delay increases the danger. Only in those cases coming to laparotomy during the first 24 to 36 hours, is it possible to relieve the condition in a few minutes after the opening of the abdominal cavity, and it is by such a rapid procedure alone that really good results are obtained, as about two-thirds of these cases are cured. When severe symptoms of incarceration are present, which take place relatively early in acute

PLATE 44.



ATRESIA OF THE INTESTINES.

I. Atresia recti vaginalis. *aa.* Bristle in opening between the rectum and vagina. *b.* Introitus vaginæ.

II. Atresia recti vesicalis. *aa.* Bristle in opening between the rectum and bladder. *b.* Openings of ureters.

III. Atresia duodeni congenita. *a.* Dilated duodenum ending in a pouch. *b.* Collapsed duodenum below blind pouch.

IV. Atresia ilei congenita ex intussusceptione intrauterina. *a.* and *b.* Two atresic portions of ileum. *c.* Intussusception. *d.* Processus vermiformis.

V. Atresia recti urethralis. *a.* Opening between the rectum and urethra. *b.* Greatly distended rectum. *c.* Openings of ureters.

invagination, and the invaginated portions are already so altered that they must be extirpated, or the adhesions formed do not permit of separation, the proportion of cures is markedly lowered, whether one resects, performs enterostomy, or makes an artificial anus. For the details of the operative procedures, the text books on the Surgery of Children are to be consulted.

Intussusceptions which extend into the rectum, or are prolapsed from the anus, do not contraindicate laparotomy if they are not gangrenous. In case this is present, one can remove the portion which can be reached, and, if this does not relieve the occlusion of the intestine, an artificial anus can be made.

In chronic intestinal intussusception, attempts at reduction by means of water and air should be made; these can be frequently repeated if there are no symptoms of incarceration. When these measures do not succeed, laparotomy should be done, and the results, as has already been stated in this form, are much more favorable.

(*d*) MALFORMATIONS IN THE REGION OF RECTUM AND ANUS

The following conditions are met with:

1. *Simple atresia of the rectum or anus,* of which three types are recognized: (*a*) simple atresia of anus, in which the rectum ending in a blind sac extends to, or nearly to, the closed anus (see Fig. 25); (*b*) simple atresia of the rectum in which an anus is present, which, however, ends blindly and to which the rectum, which likewise ends in a blind sac, is joined, by means of a short bridge of tissue; (*c*) anorectal atresia, which really represents only a higher grade of the above types, in which tissue separating the anus, ending blindly, and the blind sac of the rectum is longer and broader.

2. *Atresia of anus complicated by communication with an abnormal rectum,* including (*a*) atresia recti vaginalis (opening of the rectum in the vagina, see Fig. 1 on Plate 44); (*b*) atresia recti vesicalis (opening of rectum into the bladder, see Fig. 2 on Plate 44); (*c*) atresia recti urethralis (opening of the rectum into the urethra, see Fig. 5 on Plate 44).

3. *Atresia of anus with production of fistula from rectum.* The anus is occluded, the rectum ends in a blind sac from which there extends outward a fine fistulous tract, which opens in the median line of the perineum, in the raphe of the scrotum, or in the under surface of the penis, or in the vestibule of the vulva.

The **pathologic anatomy** of this condition is evident, from the illustration made from the specimens of the Museum of the Pathological Institute, and the reader is referred to the explanations accompanying these pictures.

The **symptoms** of these malformations consist of complete or partial stoppage of the intestinal contents, according as to whether there exists

an absolute occlusion or an abnormal opening. If the outlet is sufficiently wide the condition is not dangerous to life, except in cases where the feces are received in a cavity covered with mucous membrane, and susceptible to infection, which is particularly true of the bladder, which, under these circumstances, can become the seat of severe, progressive, fatal inflammation. If there is a complete occlusion there is failure to discharge meconium, and on inspection, or digital examination, one finds the absence of the anus, or the blind termination of the anal canal after a short course. Symptoms of intestinal occlusion soon set in, in which the child succumbs in the course of a few days, if the operative relief of the stoppage is not secured.

Fig. 25.

Simple atresia of the rectum. *a*.—Blind end of the rectum. *b*.—Strand of firm tissue extending from the blind end of the rectum to the anus. *c*.—Tract opened by operation.

The **diagnosis** of this condition is relatively easy. One should never forget to inspect carefully the anal region in every newborn child; whereby, in such a case as this, it would be noticed that instead of the normal anal opening a shallow depression is present, covered by the usual skin, or, the finger introduced in the apparently normally formed anus pushes against a firm obstruction. Frequently, when the bridge of tissue between the rectal and anal blind sac is a small one, the former may be felt, particularly as it bulges down upon the finger-tip during crying, or in any increase of abdominal pressure. The results obtained through examination with a probe or in bimanual exploration, by rectum and vagina, are less accurate, since it is rarely possible in this way to take the rudimentary end of the rectum between the fingers.

When there is an abnormal outlet of the rectum it is evident from the emptying of meconium from it. One endeavors then, by probing, to determine the course and length of the channel. The opening of the rectum into the bladder, or urethra, is recognized by feces in the urine.

Treatment.—If there is a complete blocking of rectum or anus, an operation must be quickly performed, in order to avoid the dangerous consequences of intestinal occlusion. There is not such haste necessary in the difficult plastic operations, in cases of abnormal outlet of the rectum with free passage; these can be better postponed to a later period.

If the rectal blind sac is felt low down an attempt is made to unite it with the normally situated anus. If the bridge of tissue separating the rectum from anus is very wide, an artificial anus must be made. An artificial anus can first be made to satisfy the pressing indication of producing a free passage; and, from this point the level of the outlet of the rectal blind sac can be determined, and, later, the union of the rectum with the anus brought about through the perineum.

In regard to the surgical details of the operative methods, and to the plastic procedures attempted in an abnormal outlet of the rectum, reference must be had to special works.

GASTRO-INTESTINAL DISEASES OF OLDER CHILDREN

BY
PROFESSOR R. FISCHL, OF PRAGUE

TRANSLATED BY
DR. J. H. MASON KNOX, JR., BALTIMORE, MD.

THE dominating position which the gastro-intestinal tract assumes in infant pathology, and the important influence which its diseases exert upon the general condition and the other organic systems, disappear after the course of the first two years. From this time on, local affections of the alimentary tract are observed, which when they reach a certain grade of intensity, exert an influence upon the body, but which, for the most part, are limited in their effects to the intestinal tract. It is the intention, then, in this place, to treat briefly of processes belonging to this category, and begin with

(*a*) GASTRIC INDIGESTION

This originates from the ingestion of too large a quantity of nourishment or from eating indigestible food. Occurrences of this kind are observed particularly at the fruit season, after a change in the accustomed character of the food, and especially in holiday seasons, or in hospitals at the conclusion of the visiting days.

The **symptoms** begin, as a rule, suddenly, or are introduced by a short prodromal stage which is characterized by headache, restless sleep, haggard appearance and the loss of appetite. Shortly after, fever sets in, which often is high, 40° C. (104° F.) and over; the child begins to vomit, complains of feeling bad, with headache and dizziness and is constipated. On examination, the patient gives the impression of being very ill; the tongue is swollen, its dorsum thickly furred, the breath is sour, the abdomen appears somewhat distended, particularly in its upper half. The urine is voided sparingly and is often thick in consistency, or has a reddish precipitate. A convulsion may mark the onset, and, in other ways, the cerebral cortex may appear involved, for, after going to bed, the children toss restlessly about, cry out in their sleep, gnash with their teeth, and sometimes also show anomalies of respiration and of pulse. (Variations in intensity and arrhythmia.) The duration of the trouble is, for the most part, very short; after two or three days the improvement generally becomes

rapid; more seldom, the condition is more protracted and lasts, with variations in the fever, which usually is lower in the morning, during a week or even longer.

The **diagnosis** is not always easy. Where impairment in the digestive power is present, particularly in those critical periods above mentioned, the condition is, as a rule, promptly recognized; especially when it comes on quickly during health and reaches its height in a few hours, leading to severe disturbance of the general condition, with a thick deposit on the tongue and similar symptoms. There are, however, cases commencing with convulsions, particularly in nervous children, which give the impression of beginning meningitis. Moreover the gastric form of influenza produces similar symptoms; still, the fact of a prevailing epidemic, the absence of any digestive indiscretion and the simultaneous occurrence of catarrhal influenza in the family, and finally, the later involvement of brothers and sisters will generally set one on the right track. The separation, also, of such a condition from typhoid fever, particularly when it concerns a somewhat prolonged form of illness, is occasionally rather difficult, especially as the bacteriological examination of the dejecta and the serum reaction with the blood yield no positive results during the first days. The acute febrile onset, accompanied by vomiting and malaise, of the acute exanthemata will indeed for a short time make one doubtful, but only for a few days, since the eruption and the characteristic changes in the mucous membrane of the pharynx and mouth soon clear up the situation.

The **treatment** is fairly simple: emptying the stomach and intestine with absolute restriction of diet and the use of a purgative; later, in cases of continued vomiting and fever, gastric lavage and careful return to the usual nourishment soon bring about prompt healing. As a laxative, I prefer calomel, which, in these cases, is well retained, and, according to the age of the patient, is given in from .01–.04 Gm. ($\frac{1}{6}$–$\frac{1}{2}$ gr.) every two hours until the appearance of green stools; castor oil (in soup or coffee, 1 to 2 tablespoonfuls); or, a mixture of rhubarb, manna and senna (compound infusion) in suitable proportions. When a high fever and intense cerebral phenomena are present, cold packs applied to the trunk are beneficial; a warm bath given in the evening will favorably influence the night's rest.

(*b*) RECURRENT VOMITING WITH ACETONÆMIA

I make use of the name given by Giliberti to this disease, which has been the subject of thorough study in recent years, because this writer has described most vividly the character of the illness and is not prejudiced in regard to its nature. Single cases have been described in the last century. The accurate knowledge and understanding of the disease as a peculiar symptom-complex, dates from the works of a series

of American and French writers, in addition to which, lately, there is also a publication by Misch from Heubner's Clinic.

The illness manifests itself by violent vomiting which occurs in a condition of perfect health, or after short prodromata of various kinds. In children, after the first year of age, the vomiting, which can persist for several hours or even days or weeks, ceases suddenly, and is followed by no well marked convalescence because of the immediate comfortable condition of the patient. Acetone in fairly definite quantities is found in the urine during the paroxysms of vomiting, which are repeated at various intervals, and which occur occasionally throughout the whole period of childhood up to puberty. Acetone is present also before the attack, and as a rule some time after it, and the characteristic acetone odor of the expired breath is noticed in the patient. Marfan strikingly says the odor is a mixture of the aroma of chloroform and acetic acid and may be so strong that it is noticed on entering the patient's room.

Boys are more frequently attacked (of thirty-four cases collected by Comby twenty-four occurred in males); the greatest frequency comes between two to eight years of life. The cases observed in infants (Rachford) I think are somewhat doubtful, and after puberty this malady is exceptional. Most of the French writers emphasize the suddenness of this illness which sets in without any warning, out of a clear sky and quickly reaches its climax, or appears after a short period of malaise consisting of headache, dizziness and nausea. Observers in other countries, however (Northrup and others), have claimed that symptoms antecedent to the intestinal disturbance, such as loss of appetite, clay-colored stools, often diarrhœa, and the like are not infrequently present, and state that these conditions may occur also in the interval between two attacks of vomiting.

The **symptom** of the vomiting remains in the foreground. It is produced without especial accompanying nausea, with little effort and copiously (projectile as in tuberculous meningitis). At the beginning, the vomitus consists of food and then becomes mucous and watery, and later, blood-stained in consequence of the straining, while a bilious content indicates, according to Giliberti, that the crisis is near its end. The children whose appetite is not particularly impaired, and who in consequence of the great loss of water, produced through the incessant vomiting, suffer greatly from thirst, retain absolutely nothing. Even a small swallow of ice-water, and every change in position brings on the act of vomiting, so that the little patients lie on their backs in bed in a state of the greatest lassitude. Their pinched features, the eyes surrounded with dark rims, the over-anxious mien, the dry tongue, and the great exhaustion, caused by the continual contractions of the stomach which finally bring up no further material, produce a pathetic picture. At the same time, the tongue appears more or less thickly coated, the

breathing is quickened and occasionally irregular (after the type of so-called acid respiration in diabetic coma, Edsall); the pulse is small, frequent, seldom retarded, although never arrhythmical. The temperature is normal, slightly or highly febrile. [Rachford has observed elevations of temperature to 40.5° C. (104.9° F.)].

The abdomen is usually sunken, scaphoid, seldom distended; it may be soft or sensitive. The stools during the whole duration of the attack are restrained, exceptionally loose. The swelling of the liver is occasionally demonstrable; moderate icterus has been described toward the end. So the attack persists, sometimes interrupted by intermissions in which the vomiting stops and more fluid can be borne for several hours, more often for three to five days, seldom for a week. In several observations, the vomiting returned after fourteen to twenty days, or only after intervals of weeks or months, or it may be after a year or more before it comes back. The attacks may be repeated frequently, as already mentioned, and the condition may persist throughout the whole period of infancy.

The examination of the scanty turbid *urine*, rich in salts, shows considerable acetone during, more often, before, and for a long time after the attacks. Indican, albumin, acetic acid and oxybutyric acid have also been shown to be present by several authors (Shaw, Valagussa and others); still these are not constant findings, while great weight is laid on all sides on the presence of acetone.

After the acute attack is over, recovery occurs with surprising rapidity. The appetite returns at once and a definite convalescence is scarcely observed.

Much uncertainty exists concerning the *cause* of this peculiar condition. The fact that the disease has been observed for the most part in well-to-do circles, and that in the parents of the patient the symptoms of so-called uric acid diathesis is often present, has caused certain authors (Comby, Valagussa, Rotch, Holt and others), to look upon it as an expression of a hidden uric acid diathesis, particularly as pains in the joints are observed, and a variation of the relation of the urea to uric acid has been found, the latter being increased during a paroxysm (Holt).

Other writers depending upon the swelling of the liver, which sometimes is present, and the presence of icterus at the conclusion of a paroxysm, assume an hepatic insufficiency of the liver as the cause, which leads to acetonuria in consequence of the deficient function of this organ.

The majority of writers confine themselves to the rather vague hypothesis of a disturbance of the intermediate metabolism, acid intoxication (Edsall), etc. For the sake of curiosity, the view of Krotkow is mentioned, who assumes a pseudomeningitis produced by disturbances of digestion.

All the observations are not uniform in character. There are, for example, cases described in which the paroxysm is ushered in by acute illness of another kind as angina, measles, diphtheria, etc., so that many variations occur both in the prodromata and the course of the disease. These facts have caused Hutinel, very properly, to assume two varieties of the disease, a primary and a secondary. In regard to the primary group, I would personally like to emphasize sharply and establish the hysterical character of the affection, as has been timidly suggested by others.

In addition to the fact that the disease appears almost exclusively among the better classes, and that a nervous heredity is often demonstrable, I would point particularly to its occurrence in brothers and sisters, as is repeatedly described (Hutinel and Marfan), and that it is analogous with the psychical contagion in chorea and similar disorders. The sudden onset without recognizable cause, the great intensity of the symptoms, suggest, likewise, this explanation, as does also the subsidence of the disease in a definite time. The lack of pronounced stigmata does not militate against such an assumption, since infantile hysteria at this age is, as a rule, mono-symptomatic. Moreover, the sudden cessation and the almost momentary return to complete health are, according to my view, indications in this direction. The occurrence of the attacks after excitement, or at the conclusion of either mental or bodily exhaustion, are in favor, likewise, of this assumption. The demonstration of abnormal secretion of uric acid salts has been repeatedly shown to be true in hysterical attacks, and, finally, I would mention in favor of my hypothesis the surprising result of certain therapeutic measures, having no influence on the etiology of the disease, as, for example, the darkening of the room (Rotch), the administration of a purge (Valagussa), the subcutaneous salt infusion (Marfan), which, according to the assertions of the writers mentioned, are able to shorten the attacks, all of which would favor an effect produced by suggestion.

It remains, however, to explain the *acetonuria*. For the most part, it has been shown to be present early in the course of the disease. It can, however, be a consequence of marked disturbances of digestion, as acetone is found in the urine in other different affections of the digestive apparatus in childhood. When it is present at the beginning it is possibly the result of some disturbance of digestion from which the child has previously suffered, and is only the expression of an exacerbation during the intercurrent attack. In order to determine this, one must examine the urine more frequently and continuously for acetone, in a number of affections in childhood.

The **diagnosis** of the condition is not always easy, particularly when it is the first attack. Broca has shown that there are cases of appendicitis which are accompanied with attacks of continuous vomit-

ing, and in which the removal of the appendix brings about permanent cure. The uselessness of dietetic and mechanical methods of treatment (irrigation of the stomach and intestine), also speak against the gastro-intestinal origin of the disease. The differential diagnosis, too, from tuberculous meningitis, is often difficult. However, the sudden onset, the continuation of vomiting, the lack of arrhythmic pulse and the retention of consciousness suggest recurrent vomiting. Moreover, the characteristic odor of the breath, and the large acetone content of the urine are valuable in this regard. Notwithstanding, the question can be very complicated, since, as in cases reported by Richardière and by Marfan, real cyclic vomiting and appendicitis may exist together, and the appendectomy, although it cures the latter, has no effect upon the former condition. In other cases, paroxysmal pains, the evidence of a mass at the characteristic point, or increased resistance of the muscles at the right side of the abdomen, and sensations in the neighborhood of MacBurney's point, are in favor of disease in the appendix. The diagnosis is easy in subsequent attacks.

The **prognosis** of the disease is generally very favorable; still there are in the literature several fatal cases (cited by Northrup), which warn one to have a certain reserve. On autopsy, the internal organs were found normal; only in two cases was there hypertrophy of the epithelium of the whole intestinal tract (?) and a slight degeneration of the glomeruli, findings much in need of verification.

The **treatment** should, in the first place, curtail the attack as much as possible, and, secondly, prevent its return. The most diverse methods have been used and poor results obtained. In accordance with the existing hypothesis, treatment with alkalies has been attempted (Edsall, Pierson and others); that is the administration of sodium bicarbonate or acid sodium phosphate in large doses up to 24 Gm. (360 gr.) in 24 hours, although it is not altogether clear to me how such quantities can be retained by a child which vomits every drop of fluid.* Moreover, the result obtained by me in two cases, by the means of citric acid, speaks against the necessity of large doses of alkali, from which also other authors (Griffith) saw no particular improvement.

Complete abstinence from food is obviously necessary. When the vomiting begins to abate, Marfan advises the administration of ice-cold sugar solution in order to make use of the curative effect of the carbohydrate. If the loss of fluid is great, and the appearance of the child is alarming, energetic measures are indicated, and one resorts to injection of physiological salt solution per rectum or to subcutaneous infusion in quantities of 40–60 c.c. (1–2 oz.) once or twice daily.

In the interval, the diet should be sensible and easily digestible,

*Edsall advises the administration of alkalies by rectal irrigations.

the stools regular, and mental and physical overexertion carefully avoided. Whether rendering the reaction of the urine neutral as recommended by Edsall, who administers for this purpose fixed alkalies, or the mixture suggested by Marfan [aqua destill. 1000 c.c. (1 quart) sod. sulph. 10 Gm. (2½ dr.), sod. phosph. 5 Gm. (1⅓ dr.), sod. bicarb. 5 Gm. (1⅓ dr.), sod. brom. 3 Gm. (45 gr.), a wine-glass of this before meals until the bottle is emptied, beginning every month] really prevents the onset of a new attack must be learned by further experience.

(*c*) CATARRH OF THE SMALL INTESTINE AND VOMITING AND DIARRHŒA IN OLDER CHILDREN

The increased resistance of the digestive organs in children, in their second year, is shown by the fact that disturbances of nutrition are localized in the intestine and produce symptoms which indicate that a definite portion of the alimentary tract is particularly or exclusively involved. The onset of these ailments is much less acute; they yield more readily to treatment, while severe affections of this nature, which threaten life and involve the whole organism in sympathy, seldom occur. Most frequently a transition of the disease process from the stomach to the small intestine is observed, so it seems proper to unite in a common chapter the consideration of Catarrh of the Small Intestine and Vomiting and Diarrhœa.

Etiology.—Acute catarrh of the small intestine occurs generally as a result of marked indiscretions in diet, also after the ingestion of food digested with difficulty or of doubtful quality, after overfeeding, or sudden variations in diet, such, for example, as occur in going from home to boarding-house cooking. Moreover, climatic influences play a part, as a stay on the sea-coast or at high altitudes, as do also other factors such as the use of unaccustomed products for food, the character of the drinking water and similar causes. The illness begins as a rule with fever, the height of which indicates the intensity of the digestive disturbance; at the same time there are symptoms of marked irritation of the nervous system, particularly in young and highly-strung children. Even convulsive attacks, often of great violence and long duration, may occur, but generally with the appearance of diarrhœal stools the fever quickly falls and the nervous irritation subsides. General malaise, dry mucous membranes, a tongue coated gray particularly at the base, headaches and loss of appetite are the usual accompanying symptoms. Exceptionally, colicky pains are present with rumbling noises in the abdomen preceding every evacuation, and rather numerous stools are passed containing light colored, often undigested, food particles, and such indigestible residues as fruit seeds, hulls of vegetables, etc. The movements leave the anus noisily and with a discharge of gas as though squirted from a syringe. Often the child has

not time to use a vessel. The number of evacuations usually varies between two to three and eight to ten in twenty-four hours. They follow shortly after meals, are definitely fluid, often interspersed with yellow or whitish particles and have for the most part a fecal odor. Only in prolonged cases, which have received no adequate treatment, do the stools become colorless, odorless and more frequent. At the same time, the child emaciates rapidly, becomes pale, loses sleep, voids sparingly a thick urine rich in salts, and suffers from distressing thirst.

The **treatment** of acute catarrh of the small intestine is relatively simple and generally successful. Only by the greatest neglect of all suitable measures of relief does the condition become dangerously severe, or lead to a chronic disturbance of digestion. If children about two years of age are afflicted, a strict diet in the form of weak, sweetened tea for twelve to twenty-four hours, a hot pack to the abdomen, and an astringent medicine are prescribed. For example, a thin mucilage, 1 Gm. (15 gr.) to 100 c.c. (3 oz.) water may be given containing zinc sulphate 0.1 Gm. (1.5 gr.), extract opii 0.02 Gm. ($\frac{1}{3}$ gr.) and syrup althææ 10 Gm. (2 dr.), teaspoonful every two hours (for a child at the end of its second year); or, extract hæmatoxylin 5 Gm. ($1\frac{1}{4}$ dr.) to 100 c.c. (3 oz.) water, tinct. catechu 3 Gm. (45 gr.), tinct. opii 20 to 25 drops, syrup cinnamomi 15 Gm. ($\frac{1}{2}$ oz.); a teaspoonful every two hours; or, extract columbo 2.5 to 3.5 Gm. ($\frac{2}{3}$ to 1 dr.) to 100 c.c. (3 oz.) water with tinct. opii 20 to 25 drops in equal doses, tannigen 0.25 to 0.4 Gm. (4 to 6 gr.) three times a day; tannalbin in equal doses or in the form of tannalbin chocolate, bismuthose, and similar preparations.

After the intestines have become quiet, diluted milk is given, best added to the tea; later cereal waters, and then slowly, with constant regard to the condition of the tongue, the previous normal diet is again resumed. It is advisable to continue the medication referred to for a half to a whole day after the complete cessation of the diarrhœa, as otherwise it may readily return. It goes without saying that these children, in the time immediately following, need especial supervision in the careful choice of the kind and quantity of their food, protection from taking cold, etc. In older patients, those somewhat more than three years of age, a liquid diet is sufficient (one consisting of thoroughly boiled cow's milk with addition of cocoa if desired, cereal coffee, etc., cereal decoctions, flour soups, fresh soft boiled eggs), together with the astringent medication already referred to, bring about in a short time a tranquil condition of the intestines, and shortly afterwards formed stools. One should wait for movements of this character before passing on to solid diet, which then should be in the form of gruels (from barley, rice, oatmeal), light farinaceous foods (puddings, omelette), finely hashed white meat (chicken, squab, veal, etc.).

Not infrequently, the condition described in the previous section as gastric indigestion terminates in catarrh of the small intestine, in which case the characteristic diarrhœa follows the symptoms of the affection first mentioned. Its treatment should be conducted along similar lines.

Moreover, vomiting and diarrhœa setting in at the same time or one immediately following the other, that is a true attack of diarrhœa with vomiting (gastro-enteritis, dyspeptic diarrhœa of the American writers), is not an unusual affection in children over two years of age, but it rarely reaches a severe grade or assumes a cholera-like character. In the simple forms of this malady an initial gastric lavage is of much service; however, this procedure which is so simple in the case of infants is not so pleasant in older children, who must be held firmly by suitable assistants. The physician's fingers should be protected from the child's teeth by a metal covering. When violent gastro-intestinal symptoms, accompanied by great loss of tissue fluids and marked involvement of the whole organism are present, the therapeutic procedures advised in cholera-like vomiting and diarrhœa may be employed.

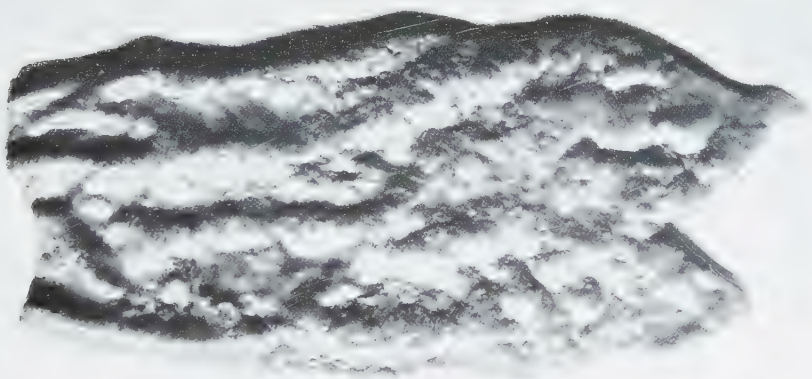
Chronic disturbances of digestion, in later childhood, have their beginnings usually in infancy, since they arise either from malnutrition at that period, or occur in consequence of a residual weakness of digestion following trifling injuries to the gastro-intestinal tract. Such children are exceedingly backward in bodily development; their dentition is delayed, they have more or less anæmia, their appetite is very variable and marked by capricious and perverse tendencies (such as the eating of chalk, dirt from the ground, etc.), their stools are at times constipated, at times diarrhœal, their tongue is always more or less coated and the abdomen distended, the region of the stomach being particularly prominent.

The *treatment* of this condition requires much patience; the most important factors are simple and not extended meals, only three a day separated by five hour intervals, a mixed diet, on the menu of which a large place should be given to special carbohydrates in the form of gruels and the vegetables, in finely divided form, which stimulate peristalsis (such as suitably prepared yellow beets, spinach, Brussels sprouts, purée of potatoes, etc.). Meat is allowed in small quantities (1 to 2 tablespoons daily) hashed, or only at one meal, and milk (and here the chief stress is to be laid) must be restricted as far as practicable, and it should be entirely withheld as a drink given with the meals. Climatic therapy is of value, such as a stay in a country region having a mild temperature, at the sea-shore, or among mountains of moderate elevation, provided that at these places food of proper quality and suitably prepared can be furnished. All excessive exercise should be avoided and the patient should remain as long as possible in the open air.

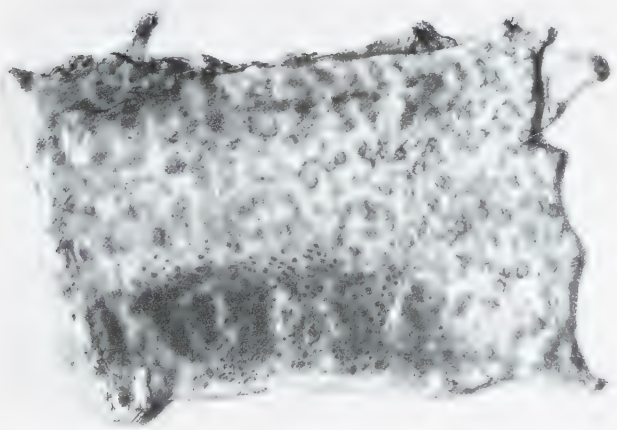
PLATE 39.

I

II



III



I. Chronic catarrh of the ileum with infiltration and pigmentation of the Peyer patches.
II. Colitis uræmica. From a case of uræmic enteritis.
III. Follicular colitis. Scars and pigmentation after inflammation of the follicles.

When anæmia is a pronounced symptom, the use of one of the numerous iron preparations will doubtless prove a valuable aid in the treatment.

(*d*) CATARRH OF THE LARGE INTESTINE IN OLDER CHILDREN

This affection can appear in acute or chronic form, since the transition of the first into the latter may occur, as well as acute exacerbations of chronic colitis. The disease begins primarily as a localized affection of the mucous membrane of the large intestine, and remains limited to this portion, or forms only the first stage of an ascending inflammation involving the whole intestinal tract; or, again, it may be the final stage of an inflammation passing from the stomach to the small intestine. Moreover it may also have a light or severe, complicated or uncomplicated, course, and out of it all results a considerable variety in the disease picture, the most marked types of which will be here presented.

Etiology.—The condition is brought about in the last analysis by indiscretions in the diet, particularly in the too free use of meat in pieces not sufficiently divided, or in giving meat too early; again, a condition of constipation is set up, after a too long continued diet of sterilized milk (Guinon). Later on, diarrhœa accompanied by a slight elevation of temperature appears with small frequent stools containing mucus and accompanied by tenesmus. The children, who are for the most part anæmic, show no particular loss in appetite and no especial disturbance of their general condition. The skin surrounding the anus is slightly reddened with superficial maceration, and one can make out, on separating the gluteal folds, a painful contraction of the sphincter ani, and transparent or light green mucus can be seen welling out of the anus. The discharge in the napkin, or in the vessel, in some cases, has an offensive or even fetid odor, and contains fecal material in the form of small solid brown particles which adhere in a gelatinous-like mass; later, it may be composed of only thick gray or green mucus, with fine flecks of blood or a diffuse tinge of blood, the evacuation of which produces pain and intense straining, so that the children cannot be removed from the vessel where even after painful straining, only a few drops of urine are pressed out. Notwithstanding the fact that the number of these (frogs' spawn-like) stools, may reach twenty to thirty in twenty-four hours, the child is not particularly emaciated by them and does not give the impression of being very ill. The anæmia, which has been present for some time, becomes more pronounced because of the considerable mixture of blood in the movements. In addition to the characteristic stools from the rectum, a fecal evacuation, coming from the upper portion of the intestinal tract, is occasionally observed after a purge or a high irrigation.

The **prognosis** of this form of colitis, arising acutely, and accompanied by moderate fever of short duration, which does not alter

markedly the condition of the patient, is favorable. The condition is seen most often in children between two and four years of age, that is at the transitional time when the child leaves its early form of nourishment and gradually takes that of adults. The disease consists of a superficial catarrh of the mucous membrane of the colon, which, in its bacteriological etiology, is not perfectly clear, but which is probably produced by the dysentery bacillus. The lesion, in most cases, involves the rectum and leads to increased secretion of its glands, hyperæmia, and cellular infiltration without extensive loss of substance.

Its **treatment** is relatively simple. An endeavor should be made to bring about a mechanical emptying of the mass of mucus collected in the large intestine, and to have in the evacuation the intestinal content of the higher portions of the alimentary tract. A form of nourishment is prescribed which limits the processes of decomposition in the intestine. Consequently, therefore, treatment should be commenced with a mild, non-irritative laxative, for example, castor oil, or powdered rhubarb, or compound liquorice powder; of the first, one tablespoonful, and of the last, as much as can be heaped upon the point of a knife. The appearance of fecal stools is usually followed by improvement and exerts a particularly favorable influence on the tenesmus. *Irrigation of the large intestine,* with a long soft rectal tube introduced high up in the bowel and slowly drawn out along the colon, is a useful local therapeutic measure. As an irrigating fluid I use, first boiled water of body temperature; when the stools contain considerable blood, 1 to 2 per cent. solution of alum, or one tenth per cent. solution of nitrate of silver (solutions of stronger concentration produce great pain), or one half per cent. solution of tannic acid (a higher content of tannin has, in some cases, been followed by symptoms of faintness suggesting collapse), or, finally, the solution of liquor alumini acetatis (P. G.) diluted one-half. Irrigations once or twice daily, according to the number of stools and the amount of mucus in the movements, are indicated, allowing $\frac{1}{2}$ to 1 quart of fluid to run in under moderate pressure. The procedure is discontinued when the tenesmus, the degree of intensity of which one can measure readily on the introduction of the rectal tube, as well as the discharge of mucus, has ceased, since by persistence of the irrigations recurrences of the affection can be easily brought about. The diet should be free from large quantities of milk, particularly in those patients who were made ill after the use of milk. Milk can be given with cocoa, cereal coffee, tapioca, or flour soup. The most suitable form of bread is zwieback; the dinner should consist of a cereal decoction, gruel (rice, barley, oatmeal, potato, grits, etc.), and a strained vegetable suitably prepared; for supper, soup made from roasted flour in which are cooked balls of dough or noodles, or to which pieces of toast are added.

Since the disease is caused by overfeeding, and since in any event a relative abstinence is of value in these mostly overfed children, one can persist in the three somewhat restricted meals. As a rule, it is possible after three or four days to introduce some variation, and to increase the amount of nourishment through eggs (omelette, soufflé, yolk of egg stirred in the soup) and by pieces of buttered zwieback; soon afterward return to normal diet by means of finely divided and thoroughly cooked meat, preserves and pastry. The meals, however, should be preferably limited to three. Constipation remaining after this condition, and in part in consequence of the curative diet, should be overcome by moderate laxatives, cold applications to the abdomen (one applied in the evening and allowed to remain until morning), as well as by the use of vegetables; as a rule, however, this constipation lasts but a short time.

Acute colitis, occurring in later childhood, does not always run its course in the benign and local manner above outlined; there are cases in which the passage of typical dysenteric stools, arising in the large intestine, last for a long time. Such children suffer from persistent constipation, pass a few hard scybala, the surface of which is often covered with mucus; their nutrition is gradually lowered, they look ill, an unpleasant odor comes from the mouth and the tongue is coated; when, suddenly, with high fever, following a very slight indiscretion in diet, and often without any recognized cause, the acute disturbance may set in with vomiting, headache, prostration and pain in the abdomen. Soon afterwards a foul smelling diarrhœa ensues which, in a short time, takes on the characteristic appearance. Hutinel, who has observed a large number of these cases, considers the condition an acute exacerbation of chronic intestinal infection localized at first in the large bowel. The affection stands in close relation to dysentery, with which idea the newer bacteriological findings mentioned elsewhere correspond. At this stage, it is still possible to accomplish much by intelligent treatment and to maintain the local character of the illness by quickly modifying the symptoms. If this opportunity is missed a number of sequelæ set in, brought about, in part, by intense intoxication; in part, by the involvement of other organs from the spread of infection from the large intestine. Among these belong cholera-like symptoms, associated with a great reduction in bodily temperature, severe nervous manifestations in the form of somnolence, slight convulsive seizures and conditions of increased irritability (certainly of purely functional nature since autopsy shows only hyperæmia of the meninges, and the spinal fluid is sterile on lumbar puncture); and, further, as an evident sign of severe intoxication there is diffuse erythema, at times resembling measles, or scarlet fever, or, again, like urticaria in appearance, with albuminuria and similar manifestations. Bronchopneu-

monia, various inflammatory conditions of the mucous membrane of the mouth and throat, multiple abscesses, vesicular eruptions, etc., should be mentioned as secondary infections. These may occur either from the entrance of micro-organisms, through the intestine primarily diseased into the lymph and blood-streams, or as a metastasis, in a body much injured in its power of resistance from other situations, skin, mouth, etc., which can often only be determined by careful post-mortem examination.

The special form of inflammation of the bladder and ascending affections of the urinary passages, occurring frequently in girls suffering from this form of infectious colitis, are to be looked upon as a continuation of the processes from the anus into the vagina and urethra.

The **treatment** of this severe form of acute inflammation of the large intestine, which shows a tendency to produce toxic and infectious complications in various parts, should be commenced as early as possible, and should aim at the complete emptying of the alimentary tract and absolute rest of the intestine. This is best accomplished by means of a diet of water, moderate purgation and intestinal irrigation. If severe vomiting is present the stomach should also be lavaged. These procedures should be continued until the odorless character of the evacuations indicates that the result desired has been accomplished. In addition, moist applications can be used to the abdomen, or, when there is great pain, opium, a few drops in mucilage, for example, for a three year old child 20 to 25 drops in 100 c.c. (3 oz.) of mucilage; of this a teaspoonful every two hours; antipyrin which Hutinel quite strongly recommends (2 to 2.5 per cent. solution), or pyramidon (.5 per cent.) which is very effective in such cases can be given. When there is high fever, and symptoms of irritation on the part of the nervous system are present, warm baths are of great service; or in such cases chloral can be given (either a teaspoonful of $\frac{1}{2}$ to 1 per cent. solution every two hours or the .25 Gm. (4 gr.) given at once). If cholera-like symptoms dominate the scene, salt infusions are indicated in addition to the application of artificial heat.

The initial return to food demands the greatest care. It is best to commence with a thin flour soup without the addition of milk or of broths, to which milk can be slowly added. After the evacuations have been normal for some time, the previous diet can be slowly resumed, in which meat particularly is deferred until later, and then in a form most easily assimilated. A tendency to constipation, which may easily result from a diet of this kind, must be met in the manner above described; where on the other hand diarrhœa is present, tannin or bismuth preparations are indicated, the latter usually combined with opium. An improvement in the diminished appetite may be brought about, as has previously been mentioned, by small doses of Carlsbad water;

I. Mucoid degeneration of the deep cells in large intestine.
II. Goblet-cell formation in epithelium of the large intestine.
III. Excessive mucoid transformation and cyst formation in large intestine.
IV. Fatty transformation and compression of gland lumen due to interstitial infiltration.
V*a* and *b*. Neighboring portion of contracted and dilated colon.

orexin tannate is of considerable use in such cases. (This is given most conveniently in the form of commercial orexin chocolates, two tablets of which can be taken an hour before meals.) The anæmia and weakness require climatic therapy assisted by the use of iron.

In addition to these acute affections of the large intestine, in older children, there are also *chronic diseases* of this portion of the alimentary tract, which occur in the form of *mucous* or *mucomembranous colitis* (Giffard, Combes and others). Children who are anæmic and who suffer from habitual constipation are, as a rule, affected, especially those who are overfed on a diet particularly rich in meat. In these children, in consequence of the fecal stasis and of the chronic irritation of the mucous membrane of the colon, an inflammatory condition is eventually set up, and this is manifested clinically by an increased production of mucus which envelops or accompanies the solid fecal masses, or, under certain conditions, by the formation of material resembling membrane. The abdomen is sunken. It is never markedly distended with gas but is sensitive along the course of the large intestine, particularly in the region of the sigmoid flexure. In sufficiently thin abdominal walls one can feel, on deep palpation, a cord-like mass resembling India rubber in consistency, which corresponds to the moderately contracted and thick-walled colon; the palpation of this mass produces considerable pain. The subjective disturbances are for the most part of a trifling nature.

The **diagnosis** is frequently only to be made after examination of the stools which consist of the products above mentioned, and which, under the microscope, are found to contain cylindrical cells, leucocytes, red blood cells, crystals of cholesterin, oxalic acid, uric acid, as well as numerous bacteria. They give the typical reaction for mucus, and the membranous type of the disease shows also shreds of fibrin.

The **treatment** of this condition consists, in the first place, in a change of diet from which meat is, as far as possible, cut out, and which should be made up for the most part of milk and cereals. (Combes has given thorough directions as to diet, which can be consulted in his book.) In addition one attempts, through mild purgation, to empty the intestine, by means of irrigations with warm water given at regular intervals, and to remove the masses of mucus and membrane resting upon the mucosa of the colon. Non-irritant fluids, such as physiological salt solution and irrigations with oil and similar materials are also useful; in addition, general treatment of a climatic nature, warm baths, massage, etc., are of service.

There are still those cases to be considered in which an acute attack of vomiting and diarrhœa or catarrh of the small intestine results in colitis. The latter condition is indicated by the appearance of mucus, pus and blood in the movements, and of tenesmus during defecation. The treatment of this condition, likewise, follows the rules above described.

(e) CONSTIPATION IN OLDER CHILDREN

It has been shown, in the discussion of Hirschsprung's disease, that in early childhood there exists a certain anatomical disposition to constipation, which has again been recently studied by Saias. He accounts for it by the relative length of the colon in proportion to that of the small intestines, the laxity of its mesentery, the winding course and the twists of the sigmoid flexure, and by the lessened development of the muscle-layers and of the elastic tissue. The hindrance of the stools from reflex causes, such as excoriations and fissures of the anus, which because of their sensitiveness in defecation, occasion retention of feces in a child, has already been mentioned. Further reasons for constipation are seen in the kind of nourishment after early infancy, and Czerny has properly called attention to the influence, in this regard, of the so-called "strengthening diet" which consists particularly of milk, meat and eggs. The foods especially suited to stimulate peristalsis, such as bread, potatoes, green vegetables, are, in this diet, wholly neglected, or given in an entirely insufficient quantity. There should be added as a further source of constipation, the wide-spread custom of giving very large quantities of cow's milk as a beverage.

Causes.—In addition to this alimentary constipation in older children, there are cases in which a hindrance to the passage of stools occurs, notwithstanding a rational diet given in proper quantities and at suitable intervals. This must be accounted for because of anatomical peculiarities in the structure of the intestine, or is due to a congenital malposition.

In school children the hurried leaving of the house, in the morning, means that they do not take a sufficient time to visit the closet, or do not remain there long enough, so that either no stool or one quite unsatisfactory is effected at the usual hour. In girls, later in life, a false sense of modesty plays a rôle at times as does the manner of dressing; and, especially in the female sex, the use of corsets acts as a restraint upon the movements of the abdomen. In addition may be mentioned the manner of living, and the sitting posture to which the school children are condemned in our present educational methods, which leads to anæmic conditions in consequence of which intestinal atrophy sets in. That catarrh of the intestinal tract, particularly colitis, can be followed by persistent constipation, has already been referred to. I would like also to point out the costive condition which may follow the exclusive milk diet after scarlet fever.

One dares not rely upon the statement of the attendant, or of the child itself, that it has a daily stool, as there are many cases who may have a passage each morning which is only partially effectual, and so they may in time suffer all the consequences of chronic constipation.

On the other hand, there are others who have no stool for a period of days or a week (the longest such interval in my personal experience concerned a child of six years and was for twelve days); and, finally, there are individuals in whom periods of normal defecation alternate with those of persistent constipation.

The *feces* appear usually very dark (as though scorched); they are dry, often covered by mucous strands, or are hard and round like goat droppings; they are evacuated during hard abdominal straining. The child plants its legs firmly on the ground, and the deeply congested face, covered with sweat, betrays the greatest exertion. Small drops or streaks of blood may be present in the movement, produced by the injury of the mucous membrane of the large intestine by the hard fecal masses. Very often it is not possible with the greatest straining to press out the scybala, and the process must be helped with the fingers.

The abdomen, in such cases, is rarely distended, so that it is possible in relaxed abdominal walls to feel the masses collected in the sigmoid flexure.

The development and health of children suffering from chronic constipation may remain for a long time undisturbed, until, quite unforseen, sudden high fever, pronounced symptoms on the part of the nervous system, particularly somnolence, even convulsions, arrhythmia of the pulse, and the like set in giving rise to the suspicion of a serious illness. I have seen one case of this kind which, after several hours of clonic contractions, profound unconsciousness, taches cérebrales, irregularity of pulse, loss of pupillary reflex,—in short, one which presented all the symptoms of tuberculous meningitis,—recovered promptly after a few doses of calomel.

Other patients exhibit anæmia of various degrees of severity, loss of appetite, different skin affections, particularly lichen urticatus, of great persistence. They complain of headache, do poor mental work, are easily fatigued, sleep restlessly, and show nervous irritability.

Indican, in considerable quantities, is found in the urine, which is often scanty and of high salt content.

The **diagnosis** of this condition usually presents no great difficulty. Hirschsprung's disease appears in earlier life and leads to the characteristic balloon-like distention of the abdomen. Chronic peritonitis is also accompanied by much more considerable meteorism, and has afterwards a fluid exudate in the abdominal cavity. Occlusion of the intestine runs its course with much more violent symptoms.

The **treatment** must first of all be directed to the cause. When the condition is due to food too rich in proteid and an oversupply of milk, a mixed diet should be given in which suitable room is reserved for carbohydrates and fats as well as for vegetables. Graham bread with butter is well taken and is of definite value, and in addition fruit

cut finely or scraped, strained vegetables, preserves not too sweet (apple jelly, plum jelly), etc.

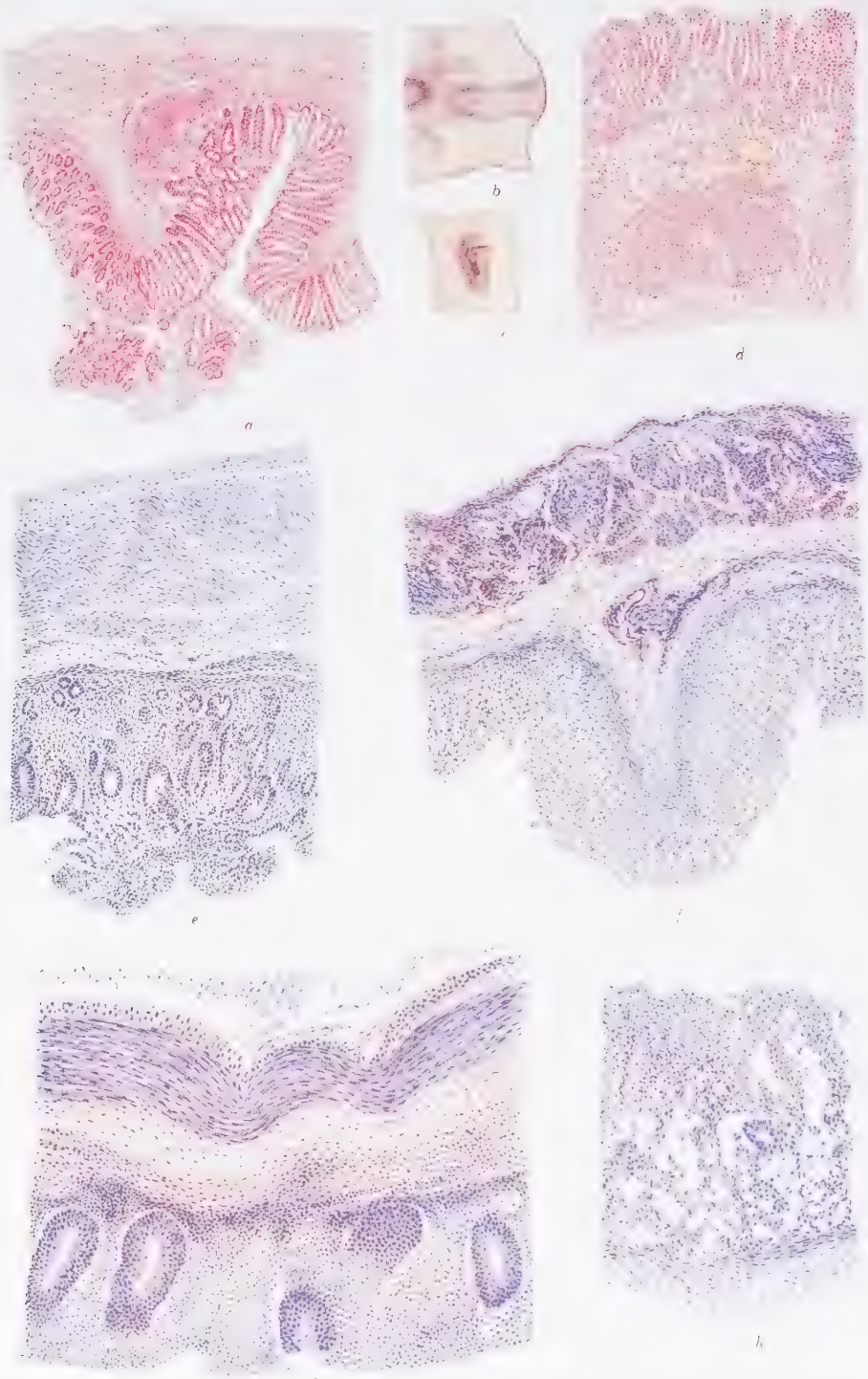
In the case of older children, who go to the closet, one must insist that this is done at an hour when they can devote sufficient time to this duty, it may be best in the evening. A moist compress to the abdomen, applied before going to sleep, assists the intestinal function. Likewise, also, massage either in the manner recommended by Heubner, in which the skin of the abdomen is taken between the index fingers and thumbs, and the abdominal wall kneaded by pinching the recti and lateral muscles of the abdomen between the fingers from above downward and from below upwards, circular movements being then made with the finger-tips between the navel and symphysis, and, finally, shoving movements, beginning at the cecum and extending along the course of the colon (each manipulation lasting two to three minutes); or in the Thure-Brand method, in which tapping movements are made with the flat of the hands in the direction of the colon, beginning at the left iliac fossa, and the rectum is kneaded between the fingers along its course, and finally the abdomen is shaken thoroughly between the hands laid on laterally (every movement takes five minutes). Intestinal irrigations, constantly reducing the temperature of the water (to about 59° F.), suppositories (glycerin, cocoa-butter, soap), small glycerin enemata (5 to 10 c.c. given with a metal syringe), etc., are all to be considered as accessory manipulations. Purgative medicines are better avoided as they do not aid the intestinal tonus and a dependence on them is easily acquired. In single cases I have had success in giving one to several tablespoonfuls of fresh butter, as suggested by Dörfler. In other instances it was not helpful. Moreover, a solution of a heaping teaspoonful of milk-sugar in a glass of water (prepared in the evening and drunk early just after awaking), is of service. When this does not suffice, mild laxatives can be employed such as syrup of figs, Seidlitz powder and similar medicines, while the drastic purges are to be avoided.

When symptoms of intestinal intoxication set in, high irrigations and the energetic use of calomel are quickly followed by improvement in the condition.

(*f*) INTESTINAL TUBERCULOSIS

Intestinal tuberculosis has been the subject of many theories and of great divergence of opinion. It is a disease, however, which by this time should be pretty thoroughly understood on account of our knowledge of the caustive agent and its pathogenic action and on account of our clinical and pathological studies.

It is not possible, in this place, to discuss even the chief features of this question and for a more extended reference the recent publications of Lesné, Finkelstein, Ibrahim and L. Fürst should be consulted.



a. Septic enteritis. Colon. Cocci over epithelium.

b. and *c*. Tuberculous ulcer in colon.

d. Stomach. Hæmorrhages in the mucosa. Lengthening of a few gland-tucts. Periarterial connective-tissue increase.

e. Stomach. Interstitial inflammation and round-cell proliferation over the epithelium.

f. Stomach in chronic gastro-enteritis.

Patholo

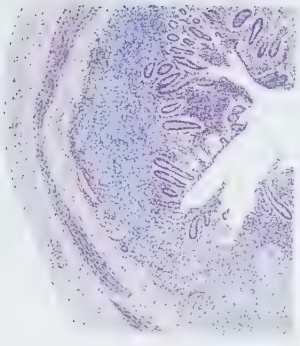
g. Large intestine. resulting in a reduction o

h. Fundus of stoma necrosis of gland-cells.

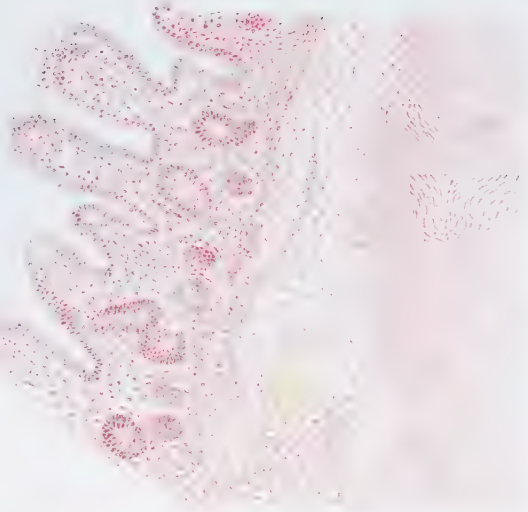
i. Colon. Chronic f

k. Ileum. Interstit

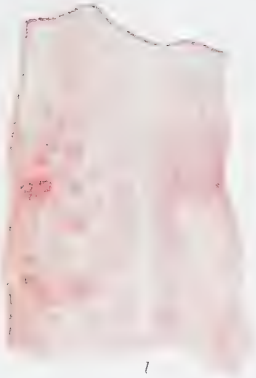
l. Large intestine.



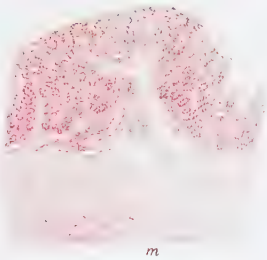
i



k



l



m

p

n

o

-enteric tract.

with interstitial infiltration,

inflammation and coagulation

on.

m. Stomach. Fundus. Dilatation of vessels and many superficial hæmorrhages in mucous membrane.

n. Megacolon congenitum. Hypertrophy of muscularis propria and muscularis mucosæ.

o. Jejunum. Marked congestion of mucous membrane.

p. Intussusceptio ileocœcalis incarcerata. Intestine removed by operation and hardened. Vertical section.

In the present article questions which are the subject of controversy will be avoided as far as possible. Numerous statistics show that primary tuberculosis of the intestinal tract, including the mucous membranes of the mouth and lymphatic glands of the throat is relatively rare in childhood. Fürst could collect only one hundred and sixty cases from the literature, which is a very small number in comparison to the vast amount of tuberculosis in the first years of life. Cases are far more common in which infection of the intestines occurs secondarily from swallowing sputum containing tubercle bacilli.

The possibility of the so-called food tuberculosis should be borne in mind, since we possess animal experiments of striking value on this subject as well as significant observations in man.

Notwithstanding the many opportunities for infection which occur in the use of food containing tubercle bacilli, the number of infections occurring in any such manner is remarkably small. Such infection may occur through the milk of tuberculous cows, dried human and bovine sputa, from a tuberculous nurse, etc.

Weleminsky claims the reason of the rarity of intestinal tuberculosis is that the tubercle bacilli present in the mouth penetrate the submaxillary and cervical lymph-nodes and thence pass into the lungs. Others believe that the action of the normal intestinal juices destroys the tubercle bacilli.

Koch and his adherents make the astonishing statement that bovine bacilli are harmless for the child, while, on the other hand, Behring and his followers assert that tuberculosis of later life has its origin in an intestinal infection acquired in early infancy and remaining latent for years. In any case, this question is unsettled and far from clear.

Milk unquestionably often contains virulent tubercle bacilli and is an important carrier of infection and open to suspicion as a means of producing the disease through the intestines.

The presence of tubercle bacilli in the secretion in the lacteal glands of phthisical wet-nurses has not been proven. At least, infections from this source are not recognized, which is not remarkable since consumptive patients are usually forbidden to nurse and especial attention is given to this condition in the engagement of a wet-nurse. Mothers suffering from acute tuberculosis expose their children to so much danger of infection in other ways that, in comparison to these, infection through the food seems relatively very improbable. In regard to the danger from the use of cow's milk or milk products (butter and cheese) containing the bovine tubercle bacilli, statistics have shown (Biedert, Ganghofner, v. Starck) that there is no relation between the frequency of pleuropneumonia in definite districts and the frequency of human tuberculosis, still less between the latter and food tuberculosis. The pathologico-anatomical studies in the various Institutes are very

different; the percentage of the involvement of primary intestinal tuberculosis in relation to that of infantile tuberculosis in general varies from 1.7 to 37 per cent. after equally careful search and the most thorough autopsy technique.

A single etiological possibility is rarely present, and it is difficult to exclude with certainty all other sources of infection; as Schlossmann has properly emphasized, a more thorough investigation in almost every case shows the possibility of infection through tuberculous patients in the neighborhood of the child.

In general, therefore, it can be stated that enterogenous infection, as far as conclusions can be drawn from clinical and pathologico-anatomical findings, is exceedingly rare in childhood, and is insignificant in comparison to the usual mode of entrance, which is the mucous membrane of the respiratory organs, and the lymphatic glands connected with it. It can not be denied that such infection can take place through the alimentary tract when it includes the cavity of the mouth and tonsils with their groups of lymph-glands, still there is need of more thorough proof founded on human material. Moreover, the view of Behring that the intestinal epithelium of young infants is permeable for bacteria, and that the invasion of the body with tubercle bacilli occurs in this manner, which, when a suitable opportunity presents itself, can be transported to distant organs, must be verified in human infants.

The *pathologic-anatomic* findings of this affection are manifold. Recent investigations have suggested that the tonsils and cervical lymph-nodes are often the seat of specific deposits; however, the tuberculous infection of these parts, or the presence in them of tubercle bacilli, is demonstrable only by means of animal experimentation. Tuberculous disease of the œsophagus is very rare, and the localization of the disease in the stomach is also uncommon either as a primary or secondary infection.

Miliary tuberculosis, which is often present in young infants, can involve the intestine and produce an eruption of tubercles, lying, for the most part, in the serous coat; but these may also be found in the mesenteric glands and peritoneum.

Chronic tuberculosis of the gastro-intestinal tract, which is of special interest, is generally localized in the mucous membrane, where it produces crater-like undermined ulcerations, at the base and border of which caseous deposits of miliary tubercles can be recognized (Figs. *b* and *c* on Plate 41). In addition there is also a decided swelling and partial caseation of the mesenteric lymph-glands. The localization of this ulcer is, for the most part, at the lowermost portion of the ileum and the region of the ileocecal valve; at those points, therefore, where the intestinal contents containing tubercle bacilli remains longest. The condition just described may be developed on the basis of a chronic

intestinal catarrh. In rare cases in children these ulcers are of considerable extent, become confluent and produce extensive loss of tissue which in certain situations leads to scar formation, which may involve the whole circumference of the intestine and so produce a stenosis in these situations.

Hypertrophic tuberculosis in the region of the cecum or of the colon, is very unusual in children; on the other hand, the appendix at this age is rather often the seat of specific inflammation, and appears, when so affected, either shriveled and hardened or swollen and lardaceous in appearance. In these cases the serosa contains caseous nodules, which are also present in the subserous tissue and in the surrounding peritoneum, while the neighboring glands undergo caseous degeneration.

The *peritoneum* is also affected in chronic tuberculosis of the intestine; single intestinal loops may be found adherent to the anterior abdominal wall, and rupture of an ulcerated portion of the intestine or of the purulent caseous content of the abdominal cavity can result. This takes place usually in the region of the navel, but can lead to the formation of abscesses or fecal fistulæ in other situations.

Isolated tuberculosis of the *mesenteric lymph-nodes,* without involvement of the adjacent portion of the intestine, is very exceptional. On the other hand, chronic tuberculous infection of the peritoneum, without tuberculosis of the intestine and with the dissemination of miliary tuberculosis, and the formation of extensive caseous deposits and masses, is of frequent occurrence.

Among the remaining abdominal organs, the liver participates in the process by a sclerotic thickening or fatty degeneration, and the spleen through chronic swelling. Other organs are seldom involved.

The **symptomatology** of this process is rather vague. Often the disease runs its course clinically under the picture of atrophy; its specific nature is guessed at from the lack of digestive indiscretions but is not definitely ascertained. Again, diarrhœa of a persistent character may set in without assignable cause, which resists all forms of medication and dietetic measures. The stools may contain bloody streaks or large quantities of blood, but in other respects exhibit no characteristic appearance. Examination of the dejecta for tubercle bacilli, which should never be omitted in suspected cases resistant to treatment, often gives the desired information.* The condition is more evident when in a child already suffering with tuberculosis of the lungs, and symptoms of collapse, violent diarrhœa suddenly sets in; the stools then show as a rule rather a large number of bacilli.

Tuberculous disease of the cecal region and the vermiform ap-

*For the demonstration of bacilli, Strassburger recommends the following procedure: Dilute the stool with water, centrifuge, decant, and treat the sediment with alcohol, again centrifuge for half a minute and prepare the specimen from the second sediment.

pendix gives rise to the clinical signs of chronic appendicitis. It may lead to abscess formation and the production of a fistula; often it runs its course quite latent and suddenly exhibits the symptoms of intestinal perforation and its sequelæ.

In general there are so few definite symptoms that, without the positive finding of the bacilli in the stools, no certain diagnosis is possible.

The **prognosis** is in general unfavorable, and the more so the younger the child. The appearance of intercurrent intestinal symptoms of a nonspecific nature hastens the end which occurs usually under the picture of chronic cachexia with slight variations in the temperature, more seldom accompanied by symptoms of intestinal perforation.

Prophylaxis is the most prominent and by all odds the most successful form of treatment. The separation of the child from its tuberculous surroundings is necessary if one would be successful, however cruel it may seem at first. The destruction of bovine tubercle bacilli by proper heating of suspected milk is a precaution which should be employed. The inoculation of cattle with tuberculin, for the purpose of disclosing latent tuberculous deposits, careful veterinary inspection, proper hygiene of the stable, control of the personnel of the dairy; in short, all the precautions recently suggested in the fight against bovine tuberculosis must be observed, since a specific therapy, such as Behring has in view, does not now exist, and immunizing procedures are not yet available.

The symptoms developing in the disease can be considerably benefited by *symptomatic therapy:* thus we use dietetic measures, medicines (tannigen, tannalbin, bismuth, opium, etc., enemata of alum water, etc.) in the treatment of diarrhœa, although in the majority of cases little is accomplished. The feeding of raw meat, recently recommended by the French authors, can be tried in the severest cases.

Where a local disease of the ileocecal region or the appendix is present an operation should be performed, and such cases offer the best outlook.

Experiences with Koch's Lymph have proved unsatisfactory, especially in intestinal tuberculosis, so that its use in this form of the disease has been entirely discontinued. Lately, Ganghofner has obtained favorable results in some cases of tuberculous peritonitis with ascites, by using minimal slowly increasing doses of old tuberculin according to the method of Goetsch. The method is worth trying.

It is to be hoped that Behring will soon keep his promise, given at the Paris Tuberculosis Congress, and place in our hands a specific remedy.

PYLORIC STENOSIS IN INFANCY

BY
PROFESSOR M. PFAUNDLER, OF MUNICH

TRANSLATED BY
DR. EDWARD J. WYNKOOP, SYRACUSE, N. Y.

INDICATIONS of a stricture in the region of the pylorus are not infrequently found among the many types of gastro-intestinal affections occurring in infancy. Often these infections are considered insignificant and are difficult of accurate determination. The symptoms of the so-called pyloric stenosis, however, should direct even the most careless observer to a narrowing or contraction of the digestive tract at the outlet of the stomach.

The collection of cases illustrating this type of disease reaches back to the 18th Century. A scientific clinical recognition of pyloric stenosis of infancy originated with a lecture given by the Danish pediatrist, Hirschsprung, before the German Pediatric Society, at Wiesbaden, in 1887. At this time Hirschsprung reported two cases which showed characteristic symptoms, the common features of which resembled "stenosis pylori congenita" in adults as reported by Maier and Landerer.

These publications of Hirschsprung stimulated others to communicate further observations. Finkelstein, in Heubner's clinic, did valuable work, which resulted in some progress in the recognition of symptoms and diagnosis. The interest in the question of pyloric stenosis in infants was aroused to the greatest extent about ten years after Hirschsprung's lecture, when the therapeutic indications became the subject of a lively controversy.

At this time the surgeons began to treat the disease. Objections were raised by the physicians against this procedure, because of a more definite knowledge about its anatomy and nature. The question now arose whether the pyloric stenosis of infancy, which had heretofore been considered exclusively a congenital organic disease, was not instead a functional disease.

And furthermore the question arose whether it were better to consider all reported observations under a common head, or under two different heads (Pfaundler, 1898). Many new authorities took part in this controversy and many new cases were used in deciding doubtful points.

If to-day one accepts the result of all these investigations, especially the purely clinical, only the dualistic standpoint is justified. Therefore of late the main interest turns to the clinical and anatomical differentiation of the organic and functional lesions and their pathogenesis; that of organic stenosis particularly being still an entirely unsolved problem. If all undoubtedly genuine tumors, which narrow the pyloric opening, the congenital atresias and strictures resulting from contracted cicatrix in early life (on account of their extraordinary rarity) are excluded, two types remain to be considered, namely, the so-called "congenital hypertrophic pyloric stenosis"* and "spasm of the pylorus."† These two types of disease stand probably in a close genetic relation to each other but practically they are very different and therefore in the following are to be separated as far as possible.

In cases of *hypertrophic pyloric stenosis,* the general aspect of the disease shows surprising uniformity, according to the description so far given. Strong and perfectly healthy children begin to vomit without recognizable cause during the first few days or weeks of life. This is the first symptom of the disease and remains continually in the fore-ground (hyperemesis lactentium). Further symptoms evidently depend on the continuous vomiting; the quantity of the stools and urine decrease, the weight curve goes rapidly down, and the child withers away. The hypogastric region is sunken in, while that of the stomach is much distended and at times moved by peristaltic waves. In severe cases, the fatal ending comes, on an average, in about six to ten weeks, with symptoms of asthenia, which conservative treatment could not stop. In other cases the objective symptoms become less pronounced, and recovery can take place in a relatively short time, seemingly under the influence of a proper regimen or else spontaneously.

The aspect of *pyloric spasm,* which is subject to much greater variations, shows some similar features, which quite often render the differentiation difficult. The differences in many cases are in degree only, yet the symptoms of the disease appear, for the most part, with digestive disturbances or accompanied by them, and intermit in a very decided way. Simple pyloric spasm rarely ends in death.

Geographical Distribution.—Hypertrophic pyloric stenosis seems to be, to some degree, peculiar to the divisions of the Anglo-Saxon race, for so far it has been described almost exclusively by German, English, American, and Scandinavian Authors. In Southern Germany and Austria it is much less prevalent than in the North. So far not one unquestioned case has been published in Latin or Slavonic Countries (May 1905). Boys outnumber the girls in the material so far known. The history of a family tendency has been repeatedly established,

*Synonyms. Constricting pyloric hypertrophy, congenital hypertrophic pyloric stenosis.
†Synonyms, pyloric spasm, congenital pyloric spasm, spasmodic stenosis.

especially among brothers and sisters. It appears very doubtful if any hereditary neuropathic tendency predisposes to the disease. A statement worthy of notice, and frequently made, is that children of parents suffering from stomach disorders are liable to this disease. More than half of all the cases so far reported were breast-fed children. Over one-third had taken women's milk exclusively. The ages of the children at the time of the occurrence of the first symptoms of the disease were as follows:

In one fourth of the cases	1- 4 days
In one fourth of the cases	4-14 days
In one fourth of the cases	2- 3 weeks
In one fourth of the cases	3- 8 weeks

Pyloric spasm in infancy occurs in different countries and seems to be widely distributed geographically. It may also occur in members of the same family. Heredity, especially a neuropathic tendency, may play a rôle in it. The first symptoms of the disease occur later as a rule, than in hypertrophic stenosis, even after the first half year of life. It attacks particularly artifically nourished children with defective digestion.

Symptoms of Hypertrophic Pyloric Stenosis.—1. *Vomiting.*—In the beginning this occurs only occasionally, then more frequently and at last after almost every feeding, either immediately, or one-half to three hours later. The quantity of matter vomited is often surprising, as it frequently exceeds the amount taken in at the last meal. The vomiting occurs suddenly and may resemble the projectile type. The vomited matter consists of food mixed with mucus which is more or less digested and at times has a very bad odor. The vomited matter is supposed never to contain bile, yet there are noteworthy exceptions to this rule. The vomiting is not accompanied by symptoms of nausea or collapse, and is not controlled by the usual remedies. The following two symptoms result from the vomiting.

2. *Constipation.*—The amount and number of the bowel evacuations are greatly reduced in some cases. Instances have been observed where no stools occured for from three to twelve days. In cases of unquestioned severity, the stools have the appearance of the so-called "starvation stools," containing practically no fecal matter. At times they are greenish or dark brown, or olive green, or resemble meconium, and are often bad smelling. The urine is scanty and highly colored.

3. The *general condition* of the child shows progressive atrophy, due to loss of water and fat. The weight curve goes down rapidly, and symptoms of extreme emaciation appear (senile face, sunken eyes, sunken fontanelles, withered skin, subnormal temperature, weakness in nursing, drowsiness, etc.).

4. Another symptom which is not always present but is worthy of notice is the refusal or the *fear of the child to drink.* The hungry child eagerly grasps the bottle or breast, only, with evident signs of pain, to

push it away again after the first few swallows. Forgetting its terrors, it then returns. This fear of the child to drink is frequently mistaken for anorexia.

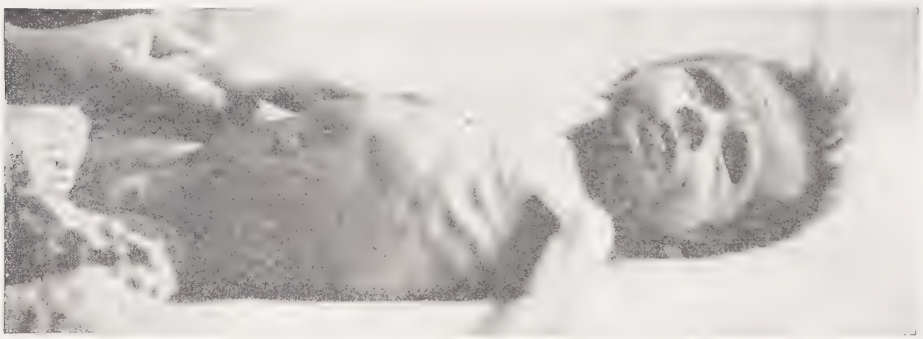
5. *Pain in the stomach* is not recognized in all cases, and is only occasionally present (pyloric spasm).

6. The *hypogastric region* is sunken in or retracted, due to the emptiness of the bowels, while the gastric region is more prominent, which is characteristic of the disease.

7. *Muscular spasm* of the stomach is easily seen through the thin abdominal walls. It is very characteristic and seldom absent. This is present in the sleeping as well as the waking child, especially toward the end of the digestive period.

The stomach becomes more prominent and peristaltic waves which resemble two balls seem to pass from left to right, and cause no pain (see Fig. 26).

Fig. 26.



Visible stomach peristalsis in hypertrophic pyloric stenosis, after a picture taken by Ibrahim.

8. *Palpable tumor* in the pyloric region (present only in the minority of cases), and even then not constant, is nevertheless significant (Finkelstein). It can be felt after the end of the fourth week of the sickness, by a slow, careful pressure through the flabby abdominal walls, in the region of the pylorus. It is characterized as a movable, resistant, tumor formation, about the size of a hazel-nut or a swollen lymph-gland. This tumor evidently corresponds to the hypertrophied pylorus, though it lies more toward the middle line than the normal pylorus, which on account of its protected position under a part of the liver is only reached with difficulty by the examining finger.

9. As a symptom of a secondary nature (2nd degree), can be mentioned *dilatation of the stomach*, which is probably present in some cases. In such a condition the lower border of the stomach may reach to or even below the navel. Whether there is simply a diminished muscular tension, an overdistention of the stomach wall or a genuine dilatation, has not as yet been determined accurately. Possibly, in the

severest cases, the three conditions might follow each other in succession. Examination shows the following disturbances of the stomach.

10. *Ischochymia.*—The effect of the motor action of the stomach in regard to the evacuation of chyme into the bowels, is very essentially diminished. Sometimes from four to five or even ten hours after taking nourishment, considerable residue remains in the stomach. This sometimes amounts to 20–50 Gm. (1–2 ounces) and can be obtained by a stomach tube, unless the stomach has been emptied by copious vomiting. The stomach contents are made up of residue from the different meals, and the stomach is seldom entirely empty, except when lavage is used. The resulting motor insufficiency is purely relative.

11. In these typical cases *hyperacidity* of the contents of the stomach was undoubtedly present, and probably also hyperchlorhydria (Freund, Ibrahim). In other cases, the acidification of the stomach contents is a normal one, which according to the conditions found in most of the other affections in infancy due to defective digestion (hyperacidity) must be striking. The effect of rennet on the stomach contents was found to be normal. The resorptive function of the mucous membrane could not be determined.

The **symptoms of pyloric spasm** are difficult to judge, owing to the meagre sources of information. The symptoms are somewhat similar, yet generally less significant. The vomiting does not occur so regularly, even stopping for days at a time. It is not so explosive in character, but more in the form of regurgitation. It comes up but little changed, shortly after taking nourishment, and at times only after overfeeding. The stools are often green, slimy, or sometimes crumbling in character. Genuine constipation alternates with diarrhœa. A slow atrophy, due to the digestive disturbance, takes place, instead of a rapid collapse. The physiological singultus is said not to be present (cardiac spasm). In cases of genuine spasm visible peristalsis undoubtedly takes place in the stomach, and perhaps also at times a tangible pyloric tumor is present. Hyperacidity or hyperchlorhydria occur usually as secondary symptoms. In these cases of pyloric spasm symptoms of a general neuropathic condition are frequently found (facial phenomena, nystagmus, myotonia).

Anatomy.—1. Children who had been suffering from the above-mentioned symptoms but who had succumbed to some intercurrent disease, failed sometimes to show at the autopsy any evidence of narrowing or any other abnormal condition at the pylorus.

2. In other cases the pylorus was found to be stiff, thick, swollen, elongated, and projecting toward the duodenum. The foundation of the clinical symptoms of congenital pyloric stenosis were frequently considered as arising from such conditions as a narrowing of the pyloric

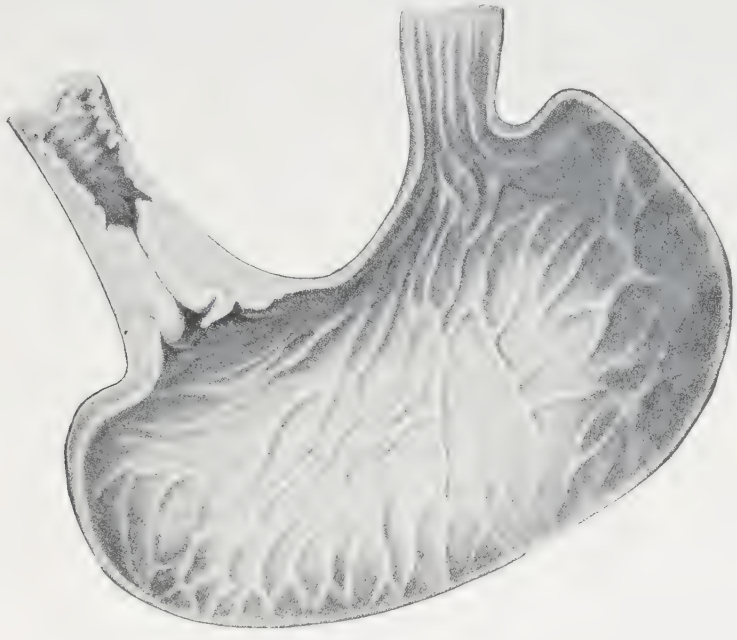
ring or the hour glass contraction of the stomach. Proper methods of measurement however, were lacking and frequently normal conditions were considered abnormal. Furthermore in view of such results, it was entirely left out of consideration, especially before 1897, just what decisive influence the condition of spasm can have upon the pylorus and shape of the stomach, even in people with healthy stomachs. The knowledge that the contracted (systolic) condition of pylorus and "antrum pyloricum," in the dead body, can simulate true hypertrophic pyloric stenosis, and very probably has done so in a number of cases, dates from that year.

3. In another group of cases, conditions were found which at first glance were different in degree only from those of the (systolic) contracted organ. Unusual thickness, elongation and cartilaginous resistance of the pylorus were found. It appears like a pipe-shaped tube between the stomach and duodenum and reaches into the latter. Longitudinal folding of the mucous membrane and effective stenosis of the pylorus are associated with this condition. The pylorus is changed to a firm walled canal about 3 cm. long, which is so extremely small that only a fine bristle can pass. No liquid can pass through this opening. A longitudinal cut through the pyloric half of the stomach reveals the fact that both "tumor" and stenosis come from a considerable thickening of the circular layer of muscles (about 3–6 mm.), in the stomach wall (4–8 mm.). Proliferation of connective tissue between the muscular tracts and a moderate increase of mucosa and submucosa are seldom found. Polypoid degeneration is also unusual.

It is now agreed that the cases in question show a true pathological change of the stomach wall and not simply a higher degree of a persistent contracted condition. In order to prove this, two things are necessary; first, that a limited elongation of the pyloric wall does not have this effect. According to all general observations, this condition relaxes the stomachs of all cadavers, even those resembling tumors, which is not the case in the simple systolic stomach. Second, that in abnormal conditions of the pylorus, not only a displacement of tissue but a positive increase of tissue, in comparison with the normal state, is present. The presentation of these two rules is considered as placing the occurrence of hypertrophic pyloric stenosis on a true anatomical foundation. In cases of hypertrophic pyloric stenosis, the rest of the stomach was found contracted in some portion and flabby in others and sometimes the muscular wall was thickened. Reliable measurements made by competent men are presented in only one case, which did not show any increase of volume (Ibrahim). Catarrhal or inflammatory changes in the mucous membrane are usually missing, though exceptionally secondary erosions, ulcers, etc., may occur. The serosa was never changed. The presence of a shortened mesentery was frequently noted.

Pathology.—The occurrence of this disease is, according to the opinion of clinicians, by no means rare, but anatomists consider pyloric stenosis in infants extremely so.

FIG. 27



Frontal cut of a stomach in a five-months-old infant that died with typical symptoms of hypertrophic pyloric stenosis.

FIG. 28.

Frontal cut through the systolic stomach of a child of eighteen months, which died of tuberculosis of the lungs, which while under observation for several months, never showed any symptoms of pyloric stenosis, misleading result of autopsy.

This shows that a good part of the cases reported have no recognizable anatomical foundation and must be considered as purely functional forms of the disease. Repeated experience has proved that under

proper treatment all symptoms of the disease may disappear temporarily, possibly, for one or two days. Negative result of autopsy on people who have died of an intercurrent disease, closes the chain of proof. In a great number of cases spastic contractions of the muscles closing the pylorus which are evidently due to certain irritations of a more or less persistent nature, are in fact cases of pyloric spasm. Functional coordinate groups of muscles might be concerned affecting different portions of the alimentary tract resulting according to location in cardiac spasm, or enterospasm.

Three different hypotheses exist at the present time with reference to the nature of stenosis and hypertrophy, as found in those cases which show an unquestioned pathological increase of tissue of the pyloric wall, namely, genuine hypertrophic pyloric stenosis.

A. The older perception points out that in congenital organic disease the presence of a genuine tumor or a tumor malformation, was the question to be considered. This view held sway up to 1897 and even since then has been frequently advocated.

The most effective argument for this view is the simultaneous occurrence of other malformations of the pylorus itself, which have been repeatedly established. Fœtal pathology however does not reveal anything that could be brought into relation to hypertrophic pyloric stenosis, and a typical finding in newborn children has so far not been made.

Two reasons have been suggested for the frequently late appearance f the first symptom in congenital stricture, viz.: (1) the relative insufficiency appears only when the stomach has been emptied entirely, and (2) that a secondary narrowing (as a wrinkling or inflammatory infiltrations of the mucous membrane) may take place. Filling the stomach with water under high pressure sometimes makes the suspected tumor entirely disappear (Freund).

B. The observation of undoubted cases of pyloric spasm in infancy, has aroused the thought that even in genuine hypertrophic pyloric stenosis such a spastic condition could have preceded. In other words that an active hypertrophy could result secondarily from the spasms.

1. It is established that spastic factors play a part also in the organic stenosis. The lumen of the pylorus evidently varies considerably as shown by sudden intermittences in the course of the disease, temporary presence of bile in the vomited food, spasmodic attacks of pain, and an incongruous relation between the clinical and anatomical findings in many cases.

2. Smooth muscle fibres are prone to functional hypertrophy. Pyloric spasm in adults leads to this condition.

3. According to an almost unanimous opinion, a far-reaching resemblance exists between the hypertrophic and the purely contracted (systolic) pylorus.

4. Evidently genuine hypertrophy is found in the same families, among brothers and sisters, as well as simple pyloric spasm. The presence of thick bands of connective tissue between the strong muscle layers, simulated through the formations of folds or oblique cuts, real overgrowth of certain parts of the mucous membrane; persistence of pyloric tumor during narcosis, and the firmer resistance of the pyloric ring against increased pressure speak perhaps against the supposition of simple persistent spasm, but not against a spasmodic hypertrophy.

SYNOPSIS OF THE MOST IMPORTANT THEORIES CONCERNING THE NATURE AND GENESIS OF HYPERTROPHIC PYLORIC STENOSIS.

1. Hypertrophy is not organic. Simulated by persistent contraction of the stomach as seen in cadavers. No increase in tissue formation. No true stenosis.	*A.* Stomach entirely normal, functions normal, during life.		
	B. Stomach remaining in systole for the same reason that an abnormal tendency to pyloric spasm existed during life (Freund).		
2. Hypertrophy is organic, due to increase in tissue formation. This is a primary congenital hypertrophy and generates stenosis and perhaps also secondary spasm.	*A.* Simple excess of new growth or tissue due to the attempt of nature to produce sufficient closure of the pylorus (Cautley, Peden).		
	B. Real tumor, leiomyoma, fibromyoma (Löbker and other surgeons).		
	C. Atavistic formation. Phylogenetic atavism (Flynn, Murray).		
	D. Ontogenetic atavism, as to the conduct in early fœtal life (Ibrahim). Disturbance of development according to Torkel.		
3. Hypertrophy is organic but is secondary to some disease originating in fœtal or extra-uterine life.	*A.* Active hypertrophy as a result of primary spasm. (Thomson).	Primary spasm	Reflex spasm of the mucous membrane of the stomach due to erosion. fissure, dyspepsia (Pritchard). Hyperchlorhydria (Knöpfelmacher). Reflex irritations from phimosis (Biedert). Hereditary nervous disposition (Freund). Abnormal formation in fœtal life of nerve apparatus making the muscle functions work antagonistically instead of synergistically (Thomson). As a result of the failure of the stomach to aid in the function of digestion.
	B. Compensatory hypertrophy after stenosis and that:		
	a. As a result of primary spastic stenosis.		
	b. As a result of primary closure of the outlet.		
	aa. Through primary organic stenosis of pylorus or folding of mucous membrane (Tilger).		
	bb. Through abnormal position, fixation. Flexion of the connecting parts of the bowels due to too short a mesentery, after fœtal inflammation (Maier, Schotten, Pitt).		

C. Inasmuch as the secondary hypertrophy concerns the pyloric ring itself less than the wall of the "antrum pyloricum," it was finally considered a compensatory hypertrophy after either a spastic or a true pyloric narrowing. The motor function of the stomach is performed almost exclusively by the walls of the "antrum pyloricum." Further hypotheses aim to show that the hypertrophy is a primary one, and that the spasm stenosis produces more marked clinical symptoms; that the stenosis had to be traced to a congenital malformation which is followed by spasm and compensatory hypertrophy; that there are congenital abnormalities of development having a very severe course, associated with a favorably progressing hypertrophy, genuine spasm, etc.

Etiology.—Nothing certain is known of the etiology of hypertrophic pyloric stenosis. In the face of its occurrence in the very young and in exclusively breast-fed children, its origin can scarcely be considered as being due to disturbances of digestion, diseases of the mucous membrane or abnormal chemical processes. In pyloric spasm such a fact could probably play a part.

Diagnosis.—The following symptoms especially are of diagnostic importance: (1) vomiting which is characteristic when it occurs without any sign of disturbed digestion in otherwise entirely healthy breast-fed children; (2) constipation especially when the lower abdomen is sunken in and the bowels appear empty; (3) visible peristalsis (not to be confounded with colon peristalsis) seen by placing the patient in a good light and applying counterirritation; (4) the presence of a pyloric tumor under light narcosis; (5) vomiting and ischochymia without hypochlorhydria.

Differential diagnostic points against hypertrophic pyloric stenosis, are to be considered:

1. The simple spasm in which all symptoms pointing to stenosis entirely disappear for a time. The constipation alternating with diarrhœa. Dyspepsia is usually present. The vomiting is less significant and the disturbance of nutrition less progressive.

2. The very rare cases of congenital atresia of the pylorus, in which the most severe symptoms appear from the first day of life and lead quickly to death.

3. The congenital occlusion and narrowing of the duodenum is regularly associated with vomiting of bile.

4. The rare cases of specific toxic effect due to mothers' or nurses' milk, in which by a change of nourishment, rapid improvement occurs (Variot).

5. The habitual regurgitation and congenital narrowing of the œsophagus, by which the nourishment is thrown up unchanged immediately after it has been taken, together with the absence of other symptoms. Pyloric stenosis will hardly be confounded with dyspepsia, peritonitis, meningitis, etc.

The **course** of the disease can show intermittent periods and resembles more the course of a more or less protracted starvation due to entire or partial abstemiousness in feeding. In fatal cases the duration of the disease depends upon the degree of stenosis and the muscular force of the stomach and must therefore vary considerably, but it is usually from 4 to 20 weeks. Healing sometimes takes place very quickly and often entirely unexpectedly, but at other times the disease may be prolonged.

In the latter case slow development of a compensatory hypertrophy of the entire muscular apparatus of the stomach may be considered possible, together with a simultaneous elongation of the lumen of the

pylorus (Hirschsprung, Ashby, Ibrahim). More or less permanent difficulties may be caused by the presence of a partial or entire stenosis continuing to an older age.

Prognosis.—In the material so far recorded, nothing is obtainable about the comparative mortality of the two forms of the disease. We can not discuss the statistics of mortality of the genuine spastic or the organic cases which have not been operated upon. The latter have so far been published to a great extent with reference to their anatomical nature. In regard to the first form only insufficient statements are at hand and a true differentiation of the two during life is often impossible.

If one counts the *mortality* of the non-operated cases, as shown by reports from the dissecting tables, as some authors do, then of course quite grotesque numbers result which have no bearing whatever upon the subject. According to the statements of the different observers the prognosis of pyloric stenosis, judged from a clinical standpoint, differs very considerably. The material varies in the different localities, especially in regard to the relative frequency of the spastic and organic form. This circumstance is not surprising in view of the geographical limitations of the hypertrophic stenosis.

Heubner says the majority of cases offer, in spite of the seriousness of the sickness, a good prognosis even under expectant treatment. Cases which show the most extreme emaciation as though the end were close at hand, sometimes make a good recovery. The longer the period between birth and the first appearance of the disease, the better the prognosis.

Treatment.—A positive diagnosis is usally not made at the beginning of the treatment. At first the disturbance is supposed to be dyspepsia with vomiting. After the proper regulation of the supply of food or a milk free diet, the vomiting does not cease, and then when the further characteristic symptoms appear, the diagnosis of pyloric spasm or hypertrophic stenosis will finally be made. At first all possible therapeutic means against spasm must be used in *every case* as it will not be possible at this time to decide which is present. Even in hypertrophic stenosis, spastic factors play an important rôle. The treatment is dietetic, physical and medicinal. These cases should be placed in hospitals where the most thorough treatment under the supervision of a resident physician and trained nurses can be carried out.

1. *Dietetics.*—If possible, breast-milk should always be used for the child's nourishment. Every endeavor must be made to keep the milk secretion active, even though the child is, at times, unable to nurse.

Even in a case where the child is able to nurse, passive feeding is to be preferred; such as giving the breast-milk in small quantities, cooled on ice, especially after vomiting, in certain cases even feeding by means of a nasal tube. The amount to be given at each feeding must be worked out in each individual case, and as soon as possible the amount

of the feeding should be increased. It may be wise to make a change, using another woman's milk in place of the mother's. If breast-milk is not obtainable, whey with 6 per cent. milk-sugar can be used in the same way. Buttermilk, cream mixtures, etc., have been recommended for the substitute feeding. Rectal feeding of milk is sometimes useful. One must not expect to keep up the normal state of the body with insufficient nourishment. Progressive loss of weight is not an immediate indication for more energetic measures.

2. *Physical Therapeutics.*—The most effective treatment is the systematic washing out of the stomach. It is surprising what good results follow this treatment which are due to the relaxing effect on the stomach wall. This has been proved experimentally. The stomach should be washed out at least twice daily with cool water, 12–16° C. (53–62° F.). The Escherich apparatus can be used. After the washing, the meal is given. Only exceptionally does the stomach washing have a bad effect on infants. Serious consequences however, can arise by the aspiration of the vomited matter, passing outside of the stomach tube, due to unskilful handling or too narrow a tube.

Furthermore, prolonged warm baths *before* the taking of nourishment and hot cataplasms to the gastric region *after*, have a favorable effect. Instead of the heavy and expensive thermophore, poultices or some other local application can be used. These do not cause any unnecessary manipulation and so do not irritate the child. Daily hypodermoclysis, or a high rectal enema of normal saline solution, is of great benefit.

Through the thin abdominal walls of cadavers of infants one can often succeed in taking hold of an œsophageal tube, which has been passed into the stomach, and guide it cautiously into the pylorus. Possibly this procedure will be used during life in cases of stenosis after the artificial relaxation of the pylorus.

3. *Medical Therapeutics.*—Opium can be used in gradually increasing doses, beginning with ½ to 1 drop of the tincture, and also atropine in very small doses. Both have a specific relaxing effect on the muscular apparatus of the gastro-intestinal tract. Alkaline drugs are of use in hyperacidity whether primary or secondary, such as magnesia, etc. Teaspoonful at meals. Bendix's formula is as follows:

R	Magn. carb.	4.0	.....	℥i
	Tinct. opii	gutt. 5-6		♏5-6
	Syr. Alth.	30.0	.....	℥i
	Aq. dest. ad	100.0		q. s. ℥iiss

Shake well and take a small teaspoonful after each meal. Purgatives are to be entirely avoided.

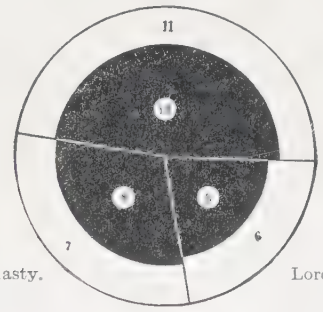
Such measures have a favorable influence not only on cases of genuine pyloric spasm, but also on cases of true organic stenosis. The results

of this treatment are of such benefit that it is to be hoped that in the future a conservative line of treatment will eliminate all other methods (Freund, Schotten).

The idea is especially promulgated among surgeons that in cases of hypertrophic pyloric stenosis, tumor formation is in question and for that reason *surgical intervention* is urged. Stern was the first to operate on such a case in 1897. The child was six weeks old and died a few hours after gastro-enterostomy. A similar fate awaited more than a dozen others who had gastro-enterostomy performed. Some eight years ago, the author of this article uttered the statement that an infant with marked emaciation and suffering from pyloric stenosis, would without doubt succumb to such a severe operation as gastro-enterostomy. This statement was not literally correct for cases have been reported which survived the operation and even, according to Ibrahim, remained entirely well (1st case of Löbker, 1898). Besides gastro-enterostomy,

Fig. 29.

Gastro-enterostomy.



Pyloroplasty. Loreta's divulsion.

Mortality chart showing results of operative treatment in hypertrophic pyloric stenosis in infants. Black—fatal result. White—recovery.

Loreta's divulsion (pyloric stretching, Nicoll, 1899), pyloroplasty (Braun, 1900), and pylorectomy (Stiles, 1900), were used. Such successful results are more often published than failures and therefore it is impossible at the present time to give definite knowledge about the real mortality of the operated cases. The chances that a cured case will be published are generally twice as great as those of one who died from an operation, so that the total mortality of cases so far published would increase from about 52 to about 65 per cent. The operative statistics therefore have at best a limited value in comparing the results of the different methods of treatment.

On referring to Fig. 29, it will be seen that treatment by Loreta's divulsion shows the best results. The poor physical condition of the infants, the difficulties of narcosis, the frequent occurrence of eventration, peritonitis, etc., explain the many unfavorable results of every operative treatment. For information with regard to technique and special reference for the indication of the different operations, one

should compare the detailed account of the different treatments of Cautley, Dent, Ibrahim, Schmidt, etc. The question whether the operative treatment of pyloric stenosis in infants appears altogether justified is answered in different ways. Surgical authors particulary, consider that after medical treatment has been carried on for some days with negative result, especially in cases of palpable pyloric tumor, further delay entails as much risk as the operation. They lay great stress upon surgical interference at the proper time. Other authors refuse from principle operative interferance (Heubner, Baginsky). Even prominent surgeons express themselves reservedly.

DISEASES OF THE APPENDIX

BY
Dr. P. SELTER, of Solingen, Germany

TRANSLATED BY
Dr. JAMES N. VANDER VEER, Albany N. Y.

Tumors of the appendix (fibroma, adenoma) occurring early in childhood have been reported in isolated cases, and show nothing characteristic at this age. Actinomycosis of this organ, in Sonnenberg's statistics, was present in only one case. Of this condition, therefore, nothing characteristic has been found in childhood.

Tuberculosis of the appendix, seldom primary, enters little into this discussion, save as general or intestinal tuberculosis, especially of the cecal region. The complaints and symptoms are as a rule the same as those of intestinal tuberculosis. Yet cases are known in which a typhlitic tumor was palpable in this region, causing pains and marked symptoms. In general, these cases are of slight practical significance. They are described under intestinal tuberculosis.

Historical.—The most common disease of the appendix is of the inflammatory type (typhlitis, peri- or epityphlitis, appendicitis, periappendicitis, skolikoiditis, periskolikoiditis). The more exact knowledge of this disease of the vermiform extension of the intestine and its surroundings, is an acquisition of the last ten years of the past century. Indeed, diseases in the region of the cecum (typhlitis stercoralis) were known to the older physicians (Aretanus, Celsus, Morgagni). There were physicians even in the first half of the past century who denied that intestinal obstruction was the chief factor of this disease, and claimed that the disease originated not in the cecum, but in the processus vermiformis itself. We have gained a more exact knowledge of the origin and course of the disease since the early and frequent operations have made possible, as it were, an autopsy during life (Sonnenberg, 1894) and from the more frequent post-mortem examinations. These confirm further the knowledge which was early established that childhood is quite often attacked by this disease (seven times more frequently than adults, Selter). This is explained by the anatomical and physiological peculiarities of infancy.

The progress of this disease in children depends on the anatomical and physiological peculiarities of childhood, as well as the organ and

its surrounding tissue. Appendicitis occurs so frequently that it should be formally designated as a disease of childhood (Sonnenberg, Karewski, Selter).

General Picture.—The disease manifests itself at the beginning, or close, of a digestive disturbance by cramps, a feeling of fulness, and pains in the right hypogastric region. There is often associated, according to the severity of the attack, nausea, vomiting, constipation or diarrhœa; a sense of pressure in the region of the appendix; swelling, pain and rigidity of the abdominal wall, or of a part thereof, especially the cecal region, and eventually a palpable tumor; many more-or-less threatening phases of suppuration and of septicæmia may appear later.

The conditions will be explained by a study of the pathological changes in the appendix and its attachments. Only in the rarest cases does the cecum show itself to be the site of the disease, and then only under entirely different circumstances; as, for instance, perforation as a result of swallowing a foreign body. Clinically, there are then presented the same symptoms as in perforation of the appendix.

Normal Anatomy.—The appendix of the child is, as a rule, located with its distal end low down in the pelvis, because of its length and the deep position of the cecum on the shelving part of the ilium. In rupture of the organ it is usually found internally and mostly in the depression between the psoas muscle and the spinal column. Only in the less frequent torsions, or where the insertion is posterior, is its course toward the lumbar region. These anatomical considerations are of great importance in determining the position of perityphlitic exudates (Selter). The younger the child, the larger and shorter in connection with the intestine is the insertion of the appendix (percentage of the length of the process to that of the larger intestine in the newborn, 1:10; in adults, 1:20, Ribbert). Scybalous masses, carriers of infection, as well as secretions can for this reason more easily enter and make their exit from the appendix.

The mucous membrane of the appendix is rich throughout in lymph-nodes and crypts. The younger the subject the richer the crypts. These crypts increase in number until they enter the submucosa.

Pathological Anatomy.—(See Plate 45.) The first signs of the disease appear in the appendiceal tonsil in the same way as we see them on the pharyngeal tonsil of children. At first there is a slight reddening and swelling of the mucous membrane, and above all, of the follicles; desquamation of the epithelium, with or without thrombosis, in the crypts or lacunæ. Otherwise there is naturally, according to the severity of the disease, a more-or-less severe lymphangitis of the wall, with œdema and œdematous swelling of the submucosa down to the serosa (appendicitis simplex, Sonnenburg).

PLATE 45.

I. Gangrenous appendicitis with perforation. Three openings, fecal concretion, circular scar, diffuse peritonitis.

II. Gangrenous appendicitis. Fecal concretions and gangrene of mucous membrane.

III. Pure perforated form. Mucous membrane, with the exception of small ulcer, is intact. Purulent fibrinous exudate on the serosa. Abscess with much pus in cul-de-sac of Douglas.

IV. Fresh suppurative appendicitis with gangrene of mucous membrane on distal end of old scar stricture. Thrombosis of veins in serosa. Surrounding tissues unaffected.

All these phases may resolve, with or without residues; may also lead to granulating processes (Riedel) and so to scar tissue and contractions caused by relapses and chronic processes. In other cases, bacteria wander into the submucous and muscular coats. The leucocytic accumulations which then arise may proceed to pus formation, and produce only the most trifling changes, except in the mucosa or serosa. Either one or both will be perforated. By perforation of the mucosa, empyema of the appendix results; and by perforation of the serosa, appendicitis perforativa (Sonnenburg). Naturally, these conditions cannot arise without leaving behind their results in the organ. More or less extensive scars, strictures, torsions, and thereby constricting granular processes remain, causing the retention of enough secretion and scybalous masses to prepare the groundwork for further inflammatory processes (Aschoff).

In more severe cases pseudomembranous lesions are found which lead to more or less deep necrosis of the mucous membrane from the lacunæ outward. The changes in the wall of the appendix already described naturally ensue. These often lead to the total destruction of that part of the wall, with perforation and severe, often pustular and inflammatory, conditions of the surrounding tissue (Aschoff).

During this change, a profuse exudation appears in the lumen of the process, according to the severity of the illness. This exudate may be expelled in milder cases. But in other cases it stagnates and is mixed with fecal material, especially when fecal matter is already present in the interior of the organ. The impregnation of the wall with inflammatory and necrotic masses is still further localized, and a higher grade of inflammation is produced at this point. The circulation in the wall is influenced by the pressure of the masses of exudate, with the formation of a thrombosis in the vessels, thus producing partial or total necrosis (appendicitis gangrænosa, Aschoff) (see Plate 45).

According to the above, the lumen of the wall of the appendix in most cases remains thickened, and commonly in other cases the serosa of the surrounding tissues participates (periappendicitis). Then, when a plug of toxine (non-bacterial) results, a fibrinous exudate about the appendix and the surrounding coils of intestine appears, or a suppuration of the bacteria-free exudate follows (peritonitis chemicalis). On the entrance, however, of bacteria-holding materials, there often occurs, according to their virulence, a more or less intense and extensive peritonitis. In particularly virulent, and also unpropitious conditions (where there are adhesions or the results of former light attacks, unsuitable retention, etc.) the whole wall will be infected in a very short time, and then the symptoms of an inflammation are evident, sometimes with slight exudation or with great quantities of fluid, and it is then

that sepsis of the peritoneum develops, which may prove fatal. In other cases there is a circumscribed inflammation which either heals, in spite of the impression it causes clinically as a large tumor, without any pus or with the formation of only trifling and quickly reabsorbed pus masses; or the inflammation may progress to the so-called *perityphlitic abscess.*

The abscess has a location varying according to the position of the processus vermiformis, and the ensuing perforation usually occurs at the distal end, either in the lesser pelvis or on the lower part of the ilium. From here it spreads toward the place of least resistance, in the lesser pelvis, and passing the lowest portion of the same, it ascends on the left side, along the rectum. If it does not enlarge, it can of itself often sink into the lesser pelvis, following the law of gravitation, and from here can rise on the left side, along the rectum. This abscess may burrow. Only in rare cases will the retrocecal position of the appendix in the lumbar region be met with. With a rapid increase of exudates, which is not a rare process in children, the above-mentioned manner of burrowing is not observed. The abscess here encounters no boundaries and soon fills a great part of the lower abdomen.

Etiology.—Diseases of the stomach and intestine frequently cause affections of the appendix in children. It is indifferent whether they are accompanied by constipation or by diarrhœa. In children we almost always find some portion of the appendix affected, chiefly in infections of the large intestine (Selter, Boas). This disease both shares in, and is classed with, practically all of those having follicular elements, for the reason that the appendix has the same anatomical structure and is of remarkable size.

It proceeds from inflammatory conditions in the cecum, conjointly with tonsillitis; it is also observed in infectious diseases, especially in large epidemic outbreaks (Sahli, Sonnenburg).

The earlier opinions which considered that a scybalous stone was in great part the cause of appendicitis and periappendicitis certainly do not hold now. The scybalous stone generally arises as a secondary factor and is etiologically of no account. Just as foreign bodies are actually drawn in—by children very frequently—they play only a secondary part in the perforation through pressure on the diseased wall of the appendix.

The same etiological importance has of late been frequently given to intestinal worms and their eggs (Renvers, Schiller). Trauma leads also, not directly but only where there exists an already diseased appendix, to perforation, torsion, or the equivalent (Sonnenberg, Payr).

There is found chiefly a mixture of various species of bacteria, with a prevailing share of the colon bacillus. Yet pure infections also appear ("Kokkenperityphlitis," Lang).

SYMPTOMATOLOGY

(*a*) LOCAL SYMPTOMS

1. Spontaneous pain in appendicitis is often complained of by older children as a feeling of heaviness, cramps or needle-like prickings. In other cases a genuine colic appears. The milder forms of periappendicitis (chronic and somewhat protracted, slowly developed or ulcerating) may also occur after severe attacks of pain; at least the same will be stated by children as being only temporary. In most cases, very severe pain is usually the warning of these attacks, and this is observed even in smaller children with slight periappendicular irritation. The severity or mildness of the pain, however, is no measure of the severity of the attack. The site of the pain is not always the cecal region but frequently will be located near the stomach. The deep position of the youthful appendix in the pelvis may result in vesical tenesmus and desire for stool.

2. Pain on pressure [tenderness] always exists in appendicitis, but varies according to the intensity of the disease. Periappendicitis is distinguished by greater sensitiveness of the appendiceal region. The skin is often hyperæsthetic over the appendix. In severe ulcerating or virulent periappendicitis, the pressure pain is extremely intense, and the abdominal wall can no longer be pressed. The pressure pain is not always situated over McBurney's point. Because of the position of the appendix in the lesser pelvis, the place of sensitiveness to pressure is frequently in another spot and is often only established through rectal palpation. All particulars as regards both kinds of pain in children are to be judged with caution. In severe cases, because of shock, or early sepsis, diminished tenderness is often present.

3. The tumor (confirmed by palpation and percussion*) is not always palpable in appendicitis. Sometimes the appendix can be palpated only as a small sensitive cord-like swelling; especially is this so in an appendix thickened by chronic processes. The appendix can be palpated as an oval or egg-shaped swelling when dilated by mucus or by pus. The previously mentioned deep position of the appendix in children, makes a bimanual examination necessary to detect its true condition. It can often be made out as a cord-like bunch in the right pelvic cavity.

Periappendicitis usually comes with a tumor-like formation which, sometimes at first, and usually after one or two days, is distinctly palpable. But a tumor is seldom clearly defined, as appendicitis is usually a limited inflammation. On the other hand very severe perforative and gangrenous forms do not reveal a circumscribed swelling,

*Palpation and percussion must be made with gentleness and exactness. In bimanual examination one finger should be in the rectum or one hand over the lumbar region and the other on the abdomen. Both sides should be compared after a thorough emptying of the bowels.

but give symptoms by an extensive board-like rigidity of the right hypogastrium or of the whole lower abdomen. Moreover, the tightly stretched prerectal peritoneal folds are not to be overlooked.

4. Distention and rigidity of the abdominal wall are found in the milder grades of appendicitis, yet the abdomen always remains compressible. Only in severe cases of pus formation in the lumen of the appendix (empyema) is there found a severe reflex rigidity in the region of the cecum. Periappendicitis is always accompanied by more or less severe distention and reflex rigidity of the abdominal musculature, or it follows surely after the first few hours or in a day, and always after a perforation of the appendix wall. It increases in undoubted and severe infectious perityphlitis, and in general periappendicitis reaches its height in the well-known board-like rigidity and distention.

Fig. 30.

Schematic illustration of the extension of a perityphilitic abscess. Observation in a six-year-old boy.

5. Disturbances of bowel movements, mostly constipation, are found in appendicitis and in periappendicitis, especially at the beginning of the disease. Previous intestinal catarrhs, which go hand in hand with perforative cases, are often first noted only as a diarrhœa. Further and favorable progress is noted by the oncoming of a normal bowel movement; and the existence or beginning of a diarrhœa or more severe constipation is looked upon as an advance of the disease process; namely, the characteristics of a perityphlitic inflammatory condition.

6. Vomiting is absent in the milder cases of appendicitis, yet it is often an early and ominous sign. In the milder forms of periappendicitis, vomiting is absent and severe gaseous eructations are almost always absent, except when they are the beginning of further progressive symptoms. At any rate the greater the participation of the abdomen in the disease, the more permanent is the vomiting; in severe cases, often after every bit of nourishment. Remission of vomiting, or its recurrence, is, as with the bowel movements, useful in the prognosis of the course of

the disease. The vomiting of dirty coffee-brown masses, stained with hæmatin clots is ominous (paralytic ileus).

(*b*) GENERAL SYMPTOMS

The general symptoms are especially important for the individual judgment of the severity of the disease and for therapeutic measures. It is not safe to draw conclusions from merely one of the general symptoms alone. Therefore the whole combination of the general symptoms, in connection with the local symptoms, is alone authorative. For instance, a rise of temperature, or an increase of the pulse rate, which often occurs, does not justify the conclusion that there is an extension of the perityphlitic inflammations, since the increase of temperature can also result from a diminution of the shock occasioned by perforation of the appendix. Concerning this last aspect of the case, the previous general symptoms, especially the blood examination and the local symptoms, give us a clue. Moreover, it is not always wise to give heed to separate symptoms, especially when taken singly.

1. The mental condition is dependent upon the severity of the infection and the cerebral anæmia which results from the congestion in the abdominal vessels, and is to be judged by the course of the remaining general symptoms.

2. The increase in body temperature is always variable in appendicitis, as well as in periappendicitis, and shows nothing characteristic. Appendicitis and periappendicitis are accompanied with low or with high temperature, which is of slight importance except that some conclusion as to the severity of the disease from this symptom can be gleaned, especially as regards a perforation. The temperature is dependent in many cases upon the shock and severity of the infection.

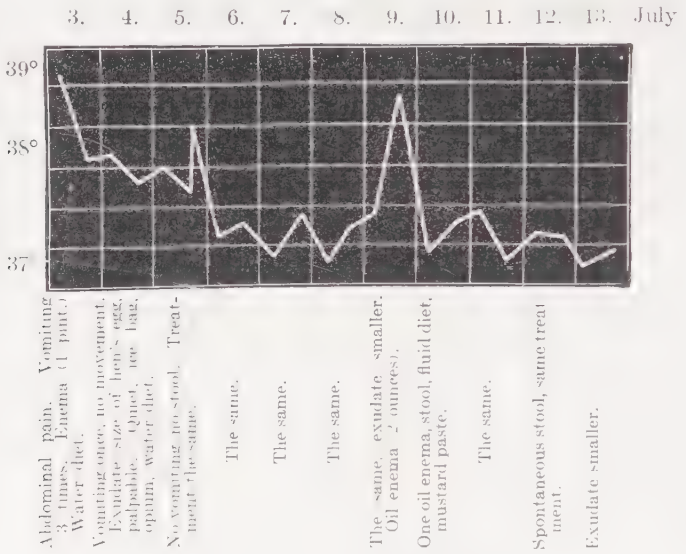
3. The behavior of the heart shows the same significance, and the quality and frequency of the pulse has significance only in conjunction with other symptoms. Small, slow or accelerated pulse in attacks of spasmodic appendicitis, or in threatened perforation, are in children often observed as the result of shock, not alone as evidence of sepsis. Smaller, more frequent or slower pulse, cyanosis, and coldness of the extremities, are not only the evidences of a more severe infection, but often show only the amount of congestion in the abdominal vessels.

4. Concerning the symptoms on the part of the blood, and especially the leucocyte count, significant observations have been made following Curschmann. In general, there is a rise of the leucocyte count to twenty or thirty thousand, per millimetre in perityphlitic suppuration. Reduction or great increase of the leucocyte count is found in acute cases of peritonitis, running a foudroyant course and usually terminating in death.

With otherwise severe symptoms, a low leucocyte count is of

ominous meaning. A rise in the leucocyte count gives hope of improvement. In the further progress of the disease, a reduction or increase of leucocytes (leucocyte curve) in conjunction with the pulse and temperature, is of value in general prognosis, and also in particular regard to the surgical aspect of the case; a falling of the leucocyte count, together with a coincident lowering of the pulse and temperature to normal, shows a favorable progress. A rise of all these curves means relapse of the inflammation; and crossing by the leucocyte curve of the rising pulse curve is unfavorable in prognosis. For differential diagnosis from ileus, as well as for confirmation of a suspected suppurative perityphlitis, the leucocyte curve is of service. For this, however, a more thorough investigation is necessary, which is difficult for

FIG. 31.


3. 4. 5. 6. 7. 8. 9. 10. 11. 12. 13. July
39°
38°
37
Abdominal pain. Vomiting 3 times. Enema (1 pint.) Water ded.
Vomiting once, no movement. Exudate size of hen's egg, palpable. Quiet, ice bag, opium, water ded.
No vomiting, no stool. Treatment the same.
The same.
The same.
The same.
The same, exudate smaller. Oil enema 2 ounces).
One oil enema, stool, fluid diet, mustard paste.
The same.
Spontaneous stool, same treatment.
Exudate smaller.

Mild appendicitis and periappendicitis

the general practitioner. For the severer cases, with undoubted implication of the abdominal wall, and threatening general perityphlitis, the leucocyte count should be subordinated because of the urgent symptoms, to the general findings otherwise observed.

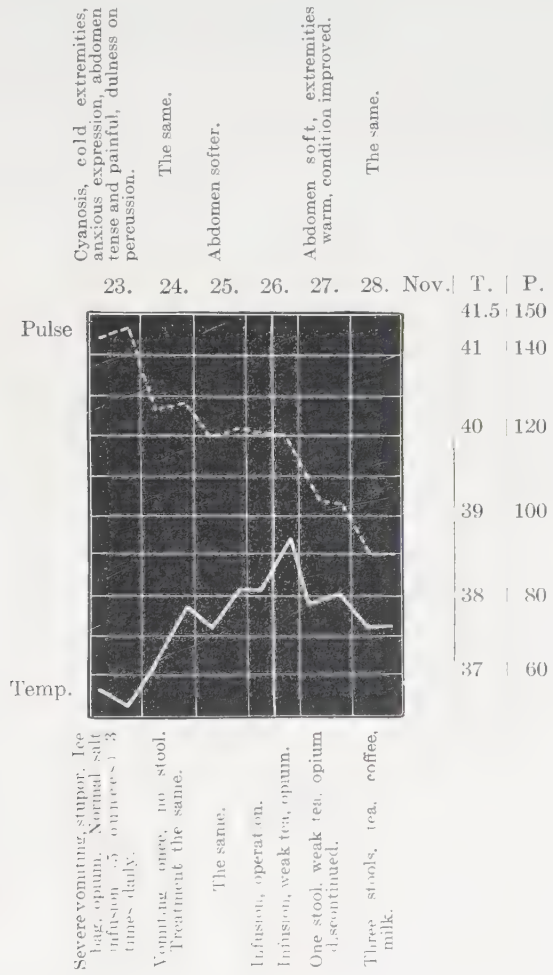
Diagnosis.—The diagnosis of the disease, after the preceding, should be clear. The trouble lies in the differentiation of appendicitis from periappendicitis, and the diagnosis of the various forms of the latter, for which our treatment is different.

Typhoid fever, ileus and perityphlitic abscesses of the burrowing form must be considered in the differential diagnosis. Exclusive from the course, the history and the leucocytosis we should consider the stools, temperature curve and the Widal reaction in the differential diagnosis from typhoid fever. The presence of a perityphilitic tumor

and examination of the hernial opening helps in a differential diagnosis with ileus. An examination of the vertebral column will exclude the presence of a psoas abscess.

Course.—The course is extremely variable. Surprises in this disease should not be unexpected. Even apparently favorable forms of appendicitis, as well as periappendicitis, often take on an

FIG. 32.


Cyanosis, cold extremities, anxious expression, abdomen tense and painful, dulness on percussion.
The same.
Abdomen softer.
Abdomen soft, extremities warm, condition improved.
The same.
23. 24. 25. 26. 27. 28. Nov.
T. | P.
Pulse
Temp.
41.5 | 150
41 | 140
40 | 120
39 | 100
38 | 80
37 | 60
Severe vomiting, stupor. Ice bag, opium. Normal salt infusion (5 ounces) 3 times daily.
Vomiting once, no stool. Treatment the same.
The same.
Infusion, operation.
Infusion, weak tea, opium.
One stool, weak tea, opium discontinued.
Three stools, tea, coffee, milk.

Severe perityphlitis with profound shock relieved by salt infusion.

unlooked for and grave course. In appendicitis, as well as in simple periappendicitis, the usual appearance after several days is as follows: The tumor becomes smaller and after a few days or weeks the pain on pressure and the sensitiveness disappear and complete recovery sets in. In other cases chronic granulating processes remain, in still others, retention of secretion in the lumen of the appendix and sometimes, torsion of the appendix occurs.

On careful examination the painful and thickened appendix can always be palpated. One attack can naturally lead to further attacks.

The second group of cases leads to suppuration in the surrounding tissue. (1) The pus formation can resolve itself, as the state of scybalous masses in old cicatrices proves. (2) In many cases a recrudescence of the symptoms occurs in the first few days, the inflammatory tumor increases in size with more pain. This can slowly heal spontaneously or become smaller or disappear more or less quickly, or burrow and become long drawn out, with fever. Even then resorption is yet possible when death from septicæmia does not follow. (3) The abscess may perforate into the intestine, the bladder, or externally. (4) The case may pass quickly into a general typhlitic condition. All these conditions may arise when the appendix has not been wisely extirpated in the beginning. In most favorable cases spontaneous healing leaves behind cicatrices, kinks, and even small abscesses which can cause further recurring relapses.

The third form of perityphlitis is even worse. The severe general symptoms with which this starts in are not relieved or seen to pass by; then immediately after the shock there ensues the appearance of a general septic inflammation. The local symptoms are exceptionally severe. The deeper situated abdominal pockets are filled with exudative masses, etc. Often a general peritonitis follows quickly or there are metastatic abscesses in the chest, phlebitis of the mesenteric veins, etc., and in a few days the child dies from septic symptoms, if a present or quickly developing peritonitis does not give a chance for life or the operative procedure does not come early enough.

Combinations of all these forms naturally occur. In by far the largest number of cases it resolves itself in children into either an appendicitis or a small periappendicitis of a mild course and with spontaneous cure; or an unfavorable course with insidious and severe purulent or putrid typhlitis. About 80 per cent. of all perityphiltic cases heal spontaneously, without recurrence, according to Baumler and Sahli. The smaller number of cases in children is in accord with this percentage for grown people. Unfortunately in the statistics of the surgeon only the severe cases clinically treated are noted, not those of private practice, so that an unprejudiced person often gets a false picture. Again, the severe pus forms, when suitably and rightly treated, make good recoveries, with a quick and correct operation.

Prophylaxis.—The prophylaxis in children is very valuable not only in the prevention of the disease, but in the avoidance of a recurrence. Evidences of digestive disturbances of all sorts, including parasites should be attended to. Careful feeding according to the age and the digestive power and regularity in bowel movement is necessary. Timely attention to the ever present digestive disturbances, especially

constipation and diarrhœa, quiet and protection as soon as the least pain comes on are among the curative measures designed to ward off appendicitis and to arrest the involvement of the peritoneum.

Therapeutics.—No firm or unbreakable rule of treatment can be laid down for the treatment of appendicitis and periappendicitis. Here, as in no other disease, is it necessary to individualize. The radical surgical element advise an unconditional early operation, and the extirpation of even slightly diseased appendices so as to offset, most optimistically, the unfortunate results of the severe symptoms in the first few days, and in the hope of spontaneous healing. The expectant treatment is important. We cannot foresee the course of the disease in the first few days. Absolute quiet, the cessation of feeding, or small sips of ice or weak tea in the milder forms (each quarter hour a teaspoonful) and the ice pack, not too heavy, fastened on a hoop stretcher, for use at night. Damp, warm compresses for the first few days are allowed as a general procedure for those who cannot endure the ice. Relative to the medical treatment, the views are widely divergent; for instance the surgical element discard the use of opium because it clouds our knowledge of the disease by placing the intestines at rest. The subcutaneous injection of morphine is recommended by some. Opium and morphine are given to children in the usual doses: tincture of opium, five times daily, one to eight drops, according to age and necessity; morphine is given with greater care, beginning with a dosage of 0.001 Gm. ($\frac{1}{60}$ gr.). In general opium should be discarded.

It is most important in all severe cases to give subcutaneous injections of normal salt solution (Friedrich) every three to four hours, 100 to 200 Gm. (3 to 6 ounces). By this means there is an increase in the number of blood corpuscles, rise of the blood pressure, irrigation of the blood, etc., and a favoring of resolution of the severe and general symptoms. Especially do the symptoms of shock and of intoxication disappear. By this method the most severe cases begin to improve under the infusion alone, and after some days the most hopeless cases sometimes assume a condition favorable for an operation. While under this treatment for one or two days a positive diagnosis and prognosis can be made. In the quickly extending perityphlitic conditions, in which the local and severe general symptoms show no abatement, one does not hesitate to operate as long as the general picture, especially the lowering of the leucocyte count, does not show a too hopeless case. From this position of operating in children during the first forty-eight hours, I have weakened because of the severe shock, and symptoms of poisoning which are present. One operates under the most favorable auspices after previous treatment with normal salt solution (compare Fig. 32).

For the remaining milder cases the expectant treatment should be

tried for the first few days. Also absolute rest, ice packs or poultices, and opium. In place of a strict hunger diet, more fluid food can be given slowly in small quantities (each quarter hour one swallow of milk). After the treatment by normal salt infusion more liquid diet by mouth can be given according as the general symptoms abate. The liquid diet should be gradually increased for some eight days; then comes a careful emptying of the bowels (oil enema) and where possible, the lightest, softest, and most easily swallowed food. The hot poultices are now applied only for a few hours in the day, the bowels are emptied daily by means of small enemas, and absolute rest should be enforced. For some weeks, with few deviations, this is the plan and general rule of conservative treatment. After some weeks most careful attention should be paid to dietetics. Surgical aid is to be sought, if at the end of the first few days a perityphlitic tumor can be palpated which increases in size, and when the symptoms are yet uncertain and the temperature does not abate.

An interval operation is indicated when small inflammatory nodules remain after the attack and a thickened appendix can be palpated bimanually and when intestinal pain is present after some months.

HERNIÆ IN CHILDREN

INGUINAL HERNIA, ABDOMINAL HERNIA, DIAPHRAGMATIC HERNIA

BY

DR. P. SELTER, OF SOLINGEN, GERMANY

TRANSLATED BY

DR. JAMES N. VANDER VEER, ALBANY, N. Y

Nature and Etiology.—Abdominal herniæ belong in general to the sphere of surgery. They deserve, however, a brief consideration in a work on children's diseases, because the characteristic hernial sacs result either from congenital conditions, errors in the fœtal development resulting in separation of the abdominal wall, or through openings or canals which have a congenitally larger dimension than normal. The child also acquires hernia from other causes, such as weakness of the tissues at certain predisposed regions, as in adult life. Hernia in children is almost always hereditary or due to congenital conditions and is therefore a disease of childhood.

This is especially true of *inguinal hernia.* This form of hernia is caused by complete or incomplete opening of the processus vaginalis peritonei (hernia vaginalis congenita s. vaginalis). This non-closure is not sufficient for the formation of the hernia. It is due to the abnormal size of the inner abdominal ring and its position directly back of and above the external abdominal ring. This gives a wide entrance, and a short, as well as direct course to the inguinal canal, a peculiarity of childhood which disappears with the growth of the child. These two characteristics, the shortness of the inguinal canal, and the location of the inner abdominal ring, together with the pockets in the infundibuliform fascia, predispose to the congenital, as well as to the acquired hernia in the child. Both kinds of inguinal herniæ are therefore attributable to an hereditary tendency. These conditions manifest the same symptoms and are differentiated only after opening the hernial sac. Congenital predisposition is also met with in other, though rare herniæ in childhood.

Of *umbilical hernia* it is not necessary to speak at length in this place.

Diaphragmatic hernia results from the partial or complete failure of the diaphragm or from abnormal enlargement of the normally existing foramina (places of predilection, the foramen of Morgagni, between

the pars sternalis and costalis; foramen Bochdaleckii, between the pars costalis and vertebralis of the diaphragm; and lastly, openings for the œsophagus or the nervus sympatheticus).

The *abdominal hernia* passes medially through congenital gaps in the linea alba or laterally through an hereditary muscular defect, or the abdominal widening of the triangle of Petit or that of Lesshaft.

The *congenital tendency* of herniæ in children indicates also the time of their occurrence. Femoral herniæ seldom occur in childhood. John Langton saw only one crural hernia among thirteen thousand observed in children. These develop in the growing pelvis and the enlarging thigh, and therefore usually appear just before or after puberty, and especially among girls. Inguinal hernia, on the contrary, is by far the most frequent in the first year of life, and almost always the indirect, which occurs from the widening of the inguinal canal, and in the majority of cases appears in boys on the right side (the right testicle descends last). Kocher never saw direct internal inguinal herniæ in children, Demme very seldom.

Umbilical herniæ observed in early childhood, or whose origin must be attributed to that period and diaphragmatic or abdominal herniæ due to congenital defects of the musculature, bring us to the conclusion that all the herniæ of childhood are traceable to *hereditary causes*. In contradistinction to the causes of hernia in adults all other causes play an unimportant part in the hernia of children. These causes are clearly a part of childhood. The distention so frequent with gastric disturbances in childhood causes a tension in the abdomen, increases the abdominal pressure, and is the chief reason for the protrusion of the viscera and expansion of the hernial sac. The crying and pressure present in these disorders have a place among the causes of herniæ not wholly imaginary. Whether phimosis and the frequent bearing down pressure play the same part seems to me by the rarity of cases of true phimosis to be uncertain.

In the literature, gastric attacks and overfeeding are not excluded. Among Jewish children hernia is not a rare but a common condition (Langton). All other causes, such as straining, and bearing down which bring about an increase of abdominal pressure, are rare in children but become more important as the child nears puberty.

The herniæ developed in childhood with the anatomical course and the etiological conditions described, do not differ in their pathologic and anatomic nature from herniæ in adults. The hernial sac and its contents are of the same character, only correspondingly more delicate and less developed.

Symptoms and Diagnosis.—The diagnosis of hernia is dependent upon a knowledge of the hernial sac, its contents and the opening. This knowledge is obtained exactly as in adults. The inguinal herniæ of

children, however, are characterized mostly by an easier reduction, as herniæ in children are almost always smaller and either make only a protuberance in the inguinal region or extend along the whole length of the inguinal canal as a sausage-shaped swelling. They seldom form large swellings in the scrotum and when present here, there is often a coil of intestine adherent. These scrotal herniæ, as those containing the cecum, or mesenteric contents, or an ovary, are frequently not reducible. The diagnosis of abdominal herniæ is the same as in adults; in diaphragmatic herniæ the sac is hard to detect.

Fig. 33.

Congenital bilateral abdominal hernia.
Congenital right inguinal hernia.

The symptoms are manifested solely from the location of the abdominal organs (stomach and intestines). Gastric disturbances, vomiting, eventual depression of the gastric region, pressure symptom from the organs of the chest, cyanosis, dyspnœa, change in the position of the heart and variations in the pulmonary note. The diagnosis of strangulation is often first made at autopsy. Disturbances of a general character are not prominent in children afflicted with herniæ, with the exception of the diaphragmatic form, because the child does not or cannot make complaint. Yet there are many nervous disorders, such as irritability, lassitude in play, as well as gastric disturbances; vomiting; eructations; irregularity of bowels, which are frequently observed when many severer and less distant symptoms have already been established.

Differential Diagnosis.—Differential diagnosis from infantile disorders, as hydrocele or delayed descent of the testicle must be made in the inguinal herniæ of children more commonly than in grown persons. Differential diagnosis from hydrocele is not always easy. Yet it can be made upon thorough examination and recognition of the following characteristics of hydrocele: failure of reposition, transparency, hollow percussion sound and the needle puncture (with a fine hypodermic syringe). In the differential diagnosis from an undescended testicle, it

is necessary to determine the absence of the testicle from its proper place, as well as the contents of the hernial sac. Abdominal herniæ do not present difficulties in differential diagnosis. It is often most difficult to diagnose a diaphragmatic hernia, and it cannot always be differentiated from other disorders.

In regard to the pathology of herniæ, fecal accumulations, inflammation and strangulation,—all these are referred to in books on surgery. Here those details are only discussed which are necessary from the general physiology and the pathology of childhood.

Progress.—Herniæ in children, with the exception of diaphragmatic hernia give a very favorable prognosis. With continued and firm retention of the hernia, and sometimes even without taking any such measure, the hernial opening contracts. The general growth of the child lengthens the inguinal canal, enlarges the swollen bowels and thickens the fatty part, and so leads to healing. Most herniæ of children heal in this way without intervention and this is especially the case with infants. Those herniæ which outlast the first year of life, and those which arise later, often have on the contrary less tendency toward cure. There always remains the question with all spontaneous reductions of herniæ, whether those afflicted with herniæ in earlier life are not in later life predisposed to recurrence.

Strangulations of herniæ are rare in children, somewhat more frequent in infants and are mostly caused by constipation.

Prognosis is favorable in the present-day asepsis and method of treatment when operated upon early, except in the very small and the very weak, or in premature children. Very large herniæ, those difficult to replace, as well as herniæ hard to keep back, can be freely operated on to-day without hesitation, when the general condition of the child will permit. Yet this is necessary in only a few cases,—according to Maass, in only one per cent. The diaphragmatic herniæ alone are an exception. Most of the children afflicted with this form die in early childhood, only a few reach a more advanced age, and are always threatened with severe disorders of nutrition, of respiration, or of circulation.

Prophylaxis.—The most important means for avoiding the formation of herniæ and for preventing their irreducibility or strangulation is to guard against or overcome the gastric attacks of children and the prevention of meteorismus due to overfeeding. All other preventive measures are of less importance or are (like the avoidance of weakness from other diseases) harder to carry out.

Treatment.—Treatment consists in replacing and holding back firmly the inguinal hernia by means of a truss covered with rubber, which fits closely to the body and does not press too tightly, and produces no eczema. Soft feathers or glycerinated absorbent cotton pads are recommended to prevent skin irritation, using one or two daily,

keeping the skin clean and well powdered, and wrapping the truss with fresh muslin each day. Instead of the truss, a dressing of woollen strands is sometimes recommended.

The injection of alcohol (Schwalbe) directly in the internal abdominal ring can be tried. For older children, an essential factor in the cure is moderate outdoor games and gymnastic exercises. These are not harmful with a well-fitting truss, on the contrary they are beneficial in so far as they strengthen the abdominal muscles. Those exercises should be selected which strengthen the abdominal muscles and cause no increase in the intra-abdominal pressure.

In the infant the struggle of trying to sit and attempting to stand (which should not be hindered but rather allowed to be tried spontaneously and for a long while) is a lively exercise for strengthening the abdominal muscles.

If the expectant treatment for one year does not bring about cure an operation should be performed and the child should not wear the truss longer than is helpful. Irreducible herniæ likewise need surgical intervention, as well as those difficult to hold back, and those in whom eczema easily develops. In strangulated herniæ surgical intervention should be undertaken without delay. Prolonged taxis, with its great dangers, should be guarded against and also the danger of narcosis in the infant. When complicated with an undescended testicle, especially when the testicle remains in the inguinal canal, it is necessary to determine absolutely whether the hernia can be replaced and the testicle can be held easily outside the inguinal canal. If such is the case a truss may be tried; but if any difficulty is experienced it is advisable to seek surgical interference. The hydrocele is best treated by puncture and injection of iodine. Yet a subsequent hæmorrhagic condition is sometimes unavoidable.

Small herniæ of the linea alba often heal spontaneously. Their treatment, which can only be surgical, is dependent upon actual trouble of the digestive organs. Lateral abdominal herniæ heal generally by closure with compresses or pads or are overcome by palliative treatment.

If there is incarceration or great difficulty of retention an operation is also indicated. For severe diaphragmatic herniæ with strangulation an operation is the only procedure. Even then the outcome is doubtful unless the lately proposed suture of the diaphragm from the pleural cavity is attempted.

ANIMAL PARASITES

BY
DR. J. LANGER, OF PRAGUE

TRANSLATED BY
DR. CHARLES F. JUDSON, PHILADELPHIA, PA.

ONLY a few years ago, worms played an important rôle in the pathology of childhood, both with physicians and the laity. The chief reason for this was the deficient knowledge, not only of many children's diseases, but also of the life conditions of these parasites. The thorough study of the latter has now completely cleared up a series of biological questions; and now, since more light has been thrown upon many obscure problems in children's diseases, a great many physicians are inclined to minimize the importance of helminthiasis in the causation of disease. The fact that in the majority of cases certain parasites call forth few or very vague symptoms when present only singly or in small number must be opposed by the other consideration that certain of our parasites are always dangerous guests, and that at times even "harmless" worms can cause serious conditions. With regard to the effect of animal parasites on their host, we must weigh the following.

1. *They withdraw albumin from their host.*—Even if this is not of much moment, as a rule, it may, nevertheless, be of importance when we have to do with a tender, youthful, and anæmic organism. According to Leuckart, the tænia saginata throws off, in the course of a year, five hundred and fifty grams of proglottides. The female ascaris produces, sometimes, forty grams of eggs in the same time. These losses in albumin, which are not essentially great, may become very much greater when a large number of parasites are present. The ankylostomum duodenale causes much more serious disturbance, since it sucks blood and can give rise to secondary hæmorrhages from the wounds it makes. Quite severe, and even fatal anæmia, may be brought about by these parasites.

2. *Their entrance, as well as their occasional wanderings through the body, may bring about a series of injuries.*—In this regard we must mention myositis from trichinæ and cysticerci, meningitis from cysticerci, liver-abscess from amœbæ and thread-worms, and fatal asphyxia due to the wanderings of the latter into the larynx and bronchial tubes.

3. *They act as foreign bodies.*—Conglomerations of round-worms have caused intestinal stenosis with fatal results. The cysticerci and

echinococci also produce many pressure symptoms and invasion symptoms, according to their size and location.

4. *They act through their products of metabolism, on the blood and the nervous system of their host.*—In latter years, the presence of blood and nerve poisons in human and animal parasites, have been demonstrated by a series of experiments. The poisonous action of echinococcus fluid has long been known. The symptomatology, diagnosis, and therapeutics of these conditions will be considered with the respective parasites.

In general, it must be emphasized that the physician should never be persuaded by the vague statements or suppositions of the parents to treat a child for worms, for those in charge often suppose that a child has worms. On the other hand, they may be easily deceived by the passage of matter resembling links, which has often been described in literature under the name of pseudohelminthiasis. An exact diagnosis is possible for most of our parasites. Only then must we order vermifuges, beginning with the milder remedies and passing to the stronger; if necessary, repeating the cure once or several times. In each case, certain questions must be taken into consideration. The following chapter will give a short résumé of that which is worth knowing in parasitology by the practitioner and the student.

I. PARASITIC PROTOZOA

The discovery of these unicellular organisms as human parasites was made several decades ago. In recent times, many case-reports have been made affirming the presence of these parasites, and the reaction of the organism which harbors them in men and animals. Experimental inoculation in suitable laboratory animals has been attempted, and positive results have been obtained. Even if up to the present time no positive evidence that these parasites give rise to intestinal disease has been furnished, still it remains positive that they find favorable conditions for existence when intestinal disease is present. It is always conceivable that they may have a part in catarrhal and inflammatory intestinal processes running a chronic course, especially when we consider their rapid and enormous powers of multiplication, their agility, and the products of their metabolism. The fact that we can not obtain pure cultures of the amœbæ, or only with great difficulty, has prevented, up to the present time, an exact demonstration of their etiological importance. The most common protozoa found in children are the following:

1. The *Amœba coli* (Fig. 34, *a*, *b*, *c*, *d*). Their appearance resembles that of the large white corpuscles, though the size of the individuals may vary considerably. In the interior of the hyalin-like body are found a nucleus; one to several strongly refractive, periodically appearing and disappearing, contractile vacuoles; and foreign bodies absorbed as

food—bacteria, plant cells, red blood-corpuscles, etc. The body is either round or oval, and is altered in shape by the extension and drawing in of the protoplasm (pseudopod formation). In the encysted condition (Fig. 34, *d*), the amœba is round. Its diameter varies between .01 and .03 mm. Quincke differentiates several forms of the amœba, some markedly pathogenic for animals and man, and some not.

Fig. 34.

Amœba coli, magnified four hundred and fifty times. Fresh preparation.

Fig. 35.

Cercomonas intestinalis, after Davaine.

Fig. 36.

Trichomonas intestinalis, magnified seven hundred times. Fresh preparation.

2. The *Cercomonas intestinalis* (Fig. 35) possesses a pearl-shaped body, which is between .008 and .012 mm. long. It has a short tail-thread, and a long flagella at the anterior end of the body.

3. The *Trichomonas intestinalis* (Fig. 36), also pear-shaped and from .01 to .015 mm. long, usually has three or four long, tender flagellæ at its anterior end and an undulating fringe of ciliæ. The tail-end is short.

Fig. 37.

Megastomum entericum, magnified thirteen hundred times; after Grassi and Schewiakoff.

Fig. 38.

Balantidium coli, magnified two hundred and fifty times.

4. The *Megastomum entericum* (Fig. 37) has a hyaline body surrounded by a delicate membrane; is from .018 to .021 mm. long; and has a breadth of .008 to .011 mm. It contains two clear vesicles, united with each other in a cavity at the anterior part of the body. Four pairs of flagellæ facilitate the movement of this parasite, which usually clings to the cells of the intestinal mucous membrane. It is a frequent parasite of mice, by which it is conveyed to food and other objects from which it gains entrance into the human body.

5. The *Balantidium coli* (Fig. 38), an egg-shaped body thickly covered with cilia, is from .05 to .10 mm. long; and contains, in addition to the food-stuffs, a nucleus and one to three contractile vacuoles. This parasite also becomes encapsulated, which, at times when permanent, admits of its conveyance by dust, water, etc. It is often found in pigs.

The **diagnosis** of these protozoa is usually easy to make with a microscope. The author recommends the immediate investigation of a small portion of feces, removed with a rectal sound, with the aid, if necessary, of a heated stage. The differential diagnosis of the individual forms offers no difficulties.

The **therapeutic** measures against these parasites are often very simple to carry out: the prevention of intestinal catarrh; the institution of a slightly constipating diet—cocoa, chocolate, and dry huckleberries or huckleberry wine, or diluted red wine. Medicines that destroy the parasites are tannalbin, 5 to 8 grains, three or four times a day; tannate of quinine, one-third to four grains (according to the age of the child), three or four times a day; calomel in small doses (one-sixth to one-third grain), three times a day; thymol (one-half to one per cent. solution) and sulphate of quinine (one to two per cent. solutions), a teaspoonful to a dessertspoonful every two hours. Intestinal irrigations after previous cleansing enemata should be made with two tenths per cent. quinine or thymol solution or one-half to one per cent. tannin solution, and are useful to destroy the amœbæ in the large colon. One should be careful not to use corrosive sublimate or carbolic acid enemata.

From the *prophylactic* standpoint, we must not forget that these parasites are usually taken into the body in an encysted condition, with water, fruit, salad, and other raw vegetables.

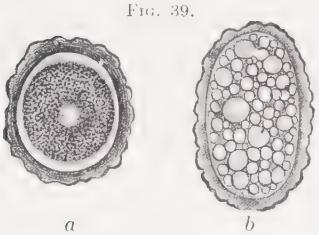
II. NEMATODES (ROUND WORMS)

1. *Ascaris lumbricoides.*—This worm has a round body, which runs to a point at both ends and is grayish red or salmon-red. Through the finely annular skin, shimmer the blood vessels of the abdomen and back, as well as a mass of twisted threads that represents the sexual organs. The mouth, which is at the anterior end of the body, has three lips with very fine teeth. The intestinal canal, which is in several sections and fills the whole body, ends at the hinder end of the body. In the male worm, which is twenty to twenty-five cm. long and three to four mm. thick, the hinder end of the body is usually slightly curved in over the abdomen; and two small, hairy spicules project from the sexual opening, which is situated in front of the anus. The sexual opening of the female, which is from thirty to forty cm. long and over five mm. thick, is at the hinder end of the anterior third of the body. According to Leuckart, the female produces from forty to sixty million eggs. The latter are .05 mm. long; have three layers, of which the outer is rough, mulberry-like, and yellowish brown; and exist in two forms (Fig. 39, *a*, *b*). According to Leuckart, the elongated oval eggs come from the virginal unfructified female. The embryos develop only from the eggs described under Fig. 39, *a*, within four to eight weeks—most

successfully when they lie superficially in moist earth, since they require oxygen. Lutz and Epstein have determined by animal experiments that round worms do not require any intermediate host. In the most manifold forms, particles of earth containing these embryo-like bodies may enter into the human body. The closer the association of the individual with the earth, the more uncleanly the person is in his whole behavior, the more frequent will be infection with these parasites. I have found, for instance, round worms present in more than seventy per cent. of Italian longshoremen; in fifty-two per cent. of Bohemian children living in the country and in only four per cent. of the children living in Prague.

The **diagnosis** of round worms is made by the laity when the worm is passed; but it is more exact to demonstrate the egg with the microscope, which is possible with the smallest portions of feces. These often adhere to the folds of the anus, and are easily obtained at any time by the introduction of a rectal sound.

Fig. 39.



a *b*

Eggs of ascaris lumbricoides.—*a*.—Round to oval. Within the outer hull a clear nucleus is usually visible in the finely granular yolk. *b*.—Elongated oval. Its contents consist chiefly of different-sized, powerfully refractive images, resembling fat droplets. These eggs are sterile, according to Lutz.

Symptoms.—Round worms can give rise to general and local symptoms. Under the former may be mentioned loss of appetite; abnormal appetite; salivation; itching of the nose; flitting abdominal pains, especially about the navel; nausea, when the stomach is empty; and diarrhœa (enteritis verminosa). A series of nervous symptoms, arrhythmia of the pulse, inequality of the pupils, urticaria, eclampsia, and choreiform and epileptiform attacks have been observed by Lutz, Mosler, and Peiper. That so manifold a symptom-complex can be due to the presence of round worms has been repeatedly thrown in doubt; but to-day it seems more intelligible to us, since the investigations of Arthus and Chanson, Von Linstow, and Nuttal have shown that ascarides may produce a poison reacting violently upon the mucous membranes, as well as upon the central nervous system.

The following **local symptoms** deserve special mention: palpable tumors, which have led to stenosis of the intestine, incarceration and volvulus, have been demonstrated to consist of a solid mass of ascarides. Every round worm may become dangerous by migration. In the stomach it will cause vomiting; in the lung, trachea, or bronchi, it will produce different degrees of suffocation, asphyxia, atelectasis, secondary pneumonia, and even death. Moreover, large numbers have been found in the biliary passages, usually of the smaller variety. There they have given rise to abscess of the liver and secondary fatal peritonitis. They have also been found as the nucleus of gall-stones. Perforation of the

healthy intestinal wall by the round worm seems very questionable, but ulcerative inflammation of the intestine may easily give rise to abscesses from which the ascarides migrate to other parts of the body.

Treatment.—A very generally employed specific against round worms is the chenopodium, the blossom of Artemisia maritima, popularly called worm-seed. The dose is 0.5 to 1 gram (7–15 grains), two or three times a day, in syrup or honey, as an electuary. The officinal preparation from this plant is the santonic acid, or santonin, of which tablets may be obtained containing from one-half to one grain.

℞ Santonin lozenges ..... 0.05 ..... gr. i
S.—For two or three days give one pill morning and evening, followed b a purge.

The latter may be given with the santonin; for example:

℞ Santonin } ..... āā 0.025 ..... gr. ss
Calomel }
Sacchari albi ..... 0.5 ..... gr. vii
S.—Give one powder morning and evening.

℞ Santonin ..... 0.10 ..... gr. iss
Olei ricini ..... 30.0 ..... f℥i
S.—Shake well. Give a teaspoonful every half-hour to one hour, until diarrhœa occurs.

Several weeks after the cure, the stools must be again examined for the eggs of the ascarides.

Prophylaxis consists in absolute cleanliness of the person, and especially of the food. Santonin must never be given on an empty stomach, and never in large doses; since, otherwise, poisoning is easily produced, with the following symptoms: general malaise, headache, vertigo, vomiting, dilated pupils, yellow vision, and convulsions. Fatal cases of santonin poisoning have been observed. After the administration of santonin, the urine becomes dark yellow and orange yellow. When dilute alkalies, caustic soda, carbonate of soda, or ammonia, are added, the urine will become a beautiful red. The treatment of santonin poisoning has for its object the removal of any unabsorbed part of the drug, by washing out the stomach and intestines and giving laxatives and emetics; and the combating of shock with black coffee, brandy, and injections of camphorated oil.

2. *Oxyuris vermicularis* (*thread-worm*).—The spindle-shaped body is white. The male is three to four mm. long and 0.1 to 0.2 mm. thick. The hinder end of the body is curved forward; the penis projects from the cloaca. The female is eight to twelve mm. long and 0.5 mm. thick. Its body is usually straight. The sexual opening is in the anterior third of the body (Fig. 40). The uterus of an adult female contains, according to Leuckart, from ten to twelve thousand eggs. These have a white color; are elongated, oval, and asymmetrical; and measure

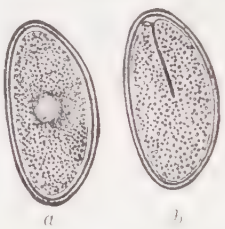
0.05 mm. long and 0.02 to 0.03 mm. broad. Within a triangular outer membrane is a finely granular yolk with a clear nucleus (Fig. 41, *a*). Often, also, they contain an embryo (Fig. 41, *b*). The oxyuris does not require an intermediate host. The embryos, after their escape from the eggs, accumulate in the small intestine, where the males and females are present in about the same number. The favorite locality for the female is the cecum, colon, and rectum. Here we find the females predominating, the males being in the ratio of only 1:10 and 1:20. The migrating female, which may be evacuated with the feces, contains many eggs and soon dies. The eggs again enter the human body by direct inoculation.

Thread-worms are distributed over the whole earth, and are met with in the best social circles. Investigation of the feces for the eggs of the oxyuris is usually, if not always, as some authors say, negative, in spite of the presence of many parasites. Only the superficial layers of the formed stool are suitable for investigating for eggs, especially

Fig. 40.

Oxyuris vermicularis. Natural size.

Fig. 41.



Eggs from oxyuris vermicularis. Magnified four hundred and twenty times.

the particles of mucus that adhere to it. One frequently finds, also, the oxyuris eggs in the dirt under the finger-nails.

Our **diagnosis** is most readily made from the visible demonstration of the oxyurides in the folds of the anus and in the genitocrural fold, or on the sound used for irrigation or for removing the feces. The evacuation after an enema usually contains several female oxyurides, which are easy to find against a black background—for example, a black rubber saucer—on account of their mobility. By giving an enema, we can nearly always make sure of our diagnosis.

Catarrhal processes in the small intestine—but more particularly in the large intestine and rectum—are set up by these parasites, if present in considerable number. Their presence in the rectum usually gives rise to intense itching and smarting, which prevents the child's falling asleep and leads to scratching and boring with the fingers,—whereby frequently, pruritis ani and secondary eczemas are produced. Oxyuris eggs have been frequently found in the scratch-marks and eczematous lesions. The irritation about the anus, and more especially the migration of the oxyurides into the vagina, can cause onanism in boys and girls

and give rise to diurnal and nocturnal eneuresis. Recently encapsulated, calcified oxyurides have been found more than once in the folds of the peritoneum in women (Kolb), probably having penetrated there from the vagina. Whether a single oxyuris, as Baginsky observes, can be the real cause of appendicitis, must still be left open to question.

In the **treatment** of oxyuriasis, always a difficult task, the physician must first of all prevent (1) the constantly recurring self-inoculation of the patient with oxyuris eggs, by the exercise of the most rigid cleanliness—repeated cleansing of the hands, with removal of the dirt under the finger-nails; sleeping with tights and gloves; and a morning bath or, at least, careful washing of the anus and genitalia. (2) The sexually ripe female worm must be removed from the rectum by injections. The usual cleansing enemata must be given daily for several weeks and medicated injection given twice a week, and always after a cleansing enema. As medicaments, one to two tablespoonfuls of vinegar, glycerin, lime-water, or salt solution may be added to one quart of water. Do not use carbolic acid or corrosive sublimate injections. (3) Internally, we can give santonin, as in the treatment of round worms; or naphthalin (according to Ungar).

℞ Naphthalini purissimi { at two years ..... 0.03 ..... gr. ss
at eight years ..... 0.15–0.2 ..... gr. iiss–iii
at twelve years ..... 0.35 ..... gr. vi
Sacchari albi ..... 0.35 ..... gr. vi
S.—Three or four such powders a day.

An eight-day interval must follow; then the cure is repeated once or twice at two weeks intervals. On account of the solubility of the naphthalin, the use of fats, butter, and oil during the naphthalin cure must be avoided; otherwise strangury may be caused. (4) The destruction of the oxyurides present in the rectum or those that have wandered out. This is accomplished by the use of mercurial suppositories or salves applied about the anus. For the latter purpose, the citrine ointment diluted half with vaseline may be used. In obstinate cases, one should investigate all the members of the family and institute treatment in all those found infected. The only *prophylaxis* is in maintaining absolute cleanliness, as is evident from our knowledge of the life-conditions of the parasite and its modes of infecting the organism.

3. The *Trichocephalus dispar* (whip-worm) is named from the thin head and neck, which are about two-thirds the length of the body. The mouth, which is at one end of the intestinal canal, has no hooklets. In the posterior third of the body are the sexual organs. The trichocephalus attaches itself very firmly to the intestinal wall, principally in the cecum, and less often in the colon or small intestine. It penetrates very deeply into the mucous membrane.

The male is three to three and a half centimetres long. The posterior part of the body is usually curved in on itself; and a hairy spicule projects from the cloaca, the sexual organ. The posterior part of the female, which is three and a half to four and a half centimetres long, is always straight. The sexual opening is in the anterior part of the thick body. The eggs, 0.05 mm. long to 0.02 mm. broad, are long and oval, and have a smooth brownish envelope and a cork-like closure at each end. The embryo develops very slowly and shows great resistance to external influences. The yolk and the developed embryo are not injured by prolonged immersion of the eggs in water; so that, besides earth, dirty water plays a part in the conveyance of this parasite.

The **diagnosis** of trichocephalus is always easy. We find the characteristic eggs, which are often associated with other worms, especially the ascaris.

The **symptoms** of the presence of trichocephalus are by no means definite. Besides a series of nervous symptoms, severe forms of enteritis have been observed when the trichocephali were present in large num-

Fig. 42.

Egg of Trichocephalus dispar, magnified 360 times. Fresh preparation.

Fig. 43.

Anchylostomum duodenale. *a*. Male. *b*. Female. Natural size.

Fig. 44.

Eggs of Anchylostomum duodenale, magnified 460 times.

bers. Of late, Girard has brought trichocephali into etiological relation with appendicitis. The parasite has the reputation of being hard to remove; but Leichtenstern mentions that he has seen individual trichocephalus worms passed after cures with extract of filix mas and thymol, in anchylostomum disease. Benzine has also been recommended. The *prophylaxis* is similar to that for round worms.

4. *Anchylostomum duodenale* (*Dochmius duodenalis*).—This parasite deserves our attention, since its presence in children and youthful persons has often been determined; though it more particularly attacks day laborers that work in mountain and tunnel excavation, quarry workers, and tillers of the soil. The male is eight to ten mm. long. The female is ten to twelve mm. long and 0.5 mm. thick (Fig. 43). The mouth has hooklike teeth; through the grayish white body, the intestinal canal, which is filled with blood, shines. The posterior part of the body of the male shows an evident broadening, from the presence of a bursa copulatrix (Fig. 43, b). The worm chiefly inhabits the duodenum and the upper ileum. The oval eggs are 0.05 to 0.06 mm. long, and 0.035 mm. broad; and are enveloped by a tender membrane. In fresh

preparations from the feces, different stages of cleavage of the yolk are usually present (Fig. 44). In a few days after the evacuation of the feces, the embryo is formed. It soon escapes into the larval stages and grows to a length of 0.5 mm. and a thickness of .02 mm. In this stage, it becomes encysted. The outer coat of the larva becomes elevated; and between this and the new skin, a hyaline globule collects. The latter protects the larva from desiccation and from the action of water. The larvæ are usually taken into the system through the drinking water. Looss and other authors have ascribed anchylostomum disease to penetration of the larvæ through the skin. Pieri doubts this.

When a considerable number of these parasites are present, signs of anæmia soon appear, following preliminary gastro-intestinal symptoms. The latter may be of different intensity and associated with various clinical symptoms. Charcot's crystals are often encountered in large number in the stools, which often contain blood.

The **diagnosis** is made sure by finding the characteristic eggs. In anæmic children of day-laborers, especially those that come from the southern parts of Europe or live there only at certain periods of the year, the stools must always be investigated for these parasites.

Treatment.—Leichenstern gives 80 grains or less of the extract of filix mas, according to the age of the child; Bozzolo recommends thymol. After-treatment is required for the anæmia, which soon improves after the parasite has been removed. A short time after the cure, the stools must again be investigated for eggs.

Prophylaxis consists chiefly in careful hygiene of the person and careful observation of the water supply. Subcutaneous infection, which may occur from bathing or wading in water, must be guarded against.

5. *Trichina spiralis.*—This thread-worm must be briefly considered here; since its presence has been discovered in children, as well as in adults. The mode of development is as follows:

The encapsulated trichinæ, male and female, enter the human stomach in trichinous pork. There they become free, and develop in the intestine into intestinal trichinæ. Two or three days after their entrance, the male and female meet; and four to five days after this, the female, which has a length of 2 to 4 mm. and a breadth of $\frac{1}{2}$ mm., deposits living embryos. According to Leuckart, the female trichina may give birth to 1500 embryos, in successive relays, in a period of five or six weeks. Some of these embryos, which are .1 mm. long and .006 mm. broad, are always evacuated with the feces; but the majority penetrate the intestinal wall and, partly by active migration and partly passively through the blood and lymph, reach the muscles, between whose fibres they develop to a size of 1 mm. and over. At the end of the third week, they roll themselves up; and by the fifth or sixth week,

become encapsulated. The capsule later becomes calcified. In this form, the muscle trichinæ may prolong their lives for years; although some of them die.

Swine are usually infected from eating rats, which in many localities are very generally infected with trichinæ. Since the rats consume the dead bodies of their own species, they constantly reinfect themselves with the parasites.

Symptoms.—Trichinosis offers the following symptoms:—In the first stage, after the importation, vague gastro-intestinal symptoms arise, with here and there diarrhœa and vomiting, and moderate fever. Eight to ten days later, general rheumatic pains develop, with high fever. The muscles are swollen and extremely painful on pressure, and also on active and passive movement. Difficulty in swallowing and attacks of dyspnœa arise. The face and the eyelids become markedly œdematous. This œdema is transitory, but often recurs. The pupils are usually fixed and dilated. Sleepiness is the rule; but exceptionally, lack of sleep is noticed in children. According to the degree of infection and the strength of the constitution of the patient, the disease ends in death or in a very protracted convalescence.

The **prognosis** is always uncertain in the beginning of the disease.

The **diagnosis** is made certain by finding the intestinal trichinæ or embryos in the evacuations; and, later, by finding muscle-trichinæ in excised portions of muscle. As a rule, the source of infection has been discovered in all epidemics.

The **treatment** consists in removing the intestinal trichinæ or embryos with laxatives (calomel, castor-oil, etc.); and, later, in destroying them with glycerin (Fiedler) or benzol (Mosler).

℞ Glycerin ..... 50.0 ..... ℥ii
S.—Several times a day, one to two teaspoonfuls.

℞ Benzol ..... 2.0–3.0 ..... gr. xxx–xlv
Mucilage of gum Arabic ..... 25.0 ..... ℥viiss
Succi liquir ..... 8.0 ..... ℥ii
Aq. menth. pip. ..... 120.0 ..... ℥iv
S.—Shake well. A teaspoonful every two hours.

In addition to careful nourishment, we must institute a symptomatic treatment: for the muscle-pains, protracted lukewarm baths, with packs and friction of chloroform liniment, etc.; in convalescence, tonic treatment. From the *prophylactic* standpoint, an obligatory and careful meat-inspection on the part of the State would accomplish very much. The most certain personal prophylaxis consists in using no pork that has not been boiled or broiled. The possibility of acquiring trichinosis from sausage, which is the most frequent source of infection, will be great in countries in which most of the rats have trichinosis.

III. CESTODES, OR TAPEWORMS

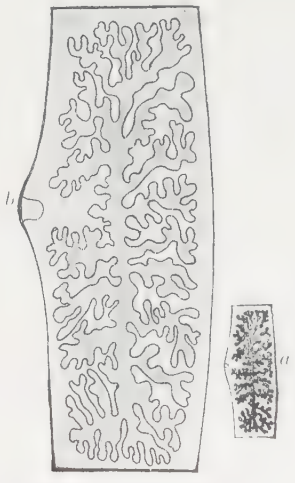
1. *Tænia solium*, the *armed tapeworm.*—This attains a length of six to nine feet. On the small head, which reaches the size of a pin-head, are four suckers and a powerful rostellum, armed with twenty-five or thirty hooks (Fig. 45). Behind the neck, which is only one centimetre long, come a long series of segments, three feet in length, becoming gradually squarer; behind these, the sexually mature segments. The latter are 9 to 10 mm. long and 6 to 7 broad.*

The uterus, which is packed full of eggs, shows on each side eight to ten dendrically branched arms, or twigs (Fig. 46). The ripe proglottides are passed in part singly, and in part with several hanging together. The eggs (Fig. 47) are round, and have a diameter of .03 mm.

Fig. 45.

Head of Tænia solium: *a*, natural size; *b*, magnified 18 times.

Fig. 46.



Single segment of Tænia solium: *a*, natural size; *b*, magnified 3½ times.

The external coat consists of delicate, radiating brownish rods, which give the appearance of a fine mosaic, under a high power (Fig. 47, *a*). On section of the egg, we see the embryo fitted out with hooklets within the lining membrane (Fig. 47, *b*).

Eating pork that contains living cysts gives rise to this disease in man. The cysticercus cellulosæ presents a small vesicle, which may reach the size of a hemp-seed; and, besides a small amount of albuminous fluid, contains the completely developed head of the Tænia solium. Swine acquire the measles by eating the proglottides, or eggs of the parasite; these animals at pasture, having plentiful opportunity to take in human excrement in meadows, woods, streets, or roads, are affected with this disease in a good deal higher proportion than are swine kept

* The measures of the links, or proglottides, of the individual tænia given are only average values.

in stalls. Man can also take in the cycticercus of the tænia solium, and this is usually due to lack of cleanliness. The insane frequently infect themselves, or the infection may come from other persons.

The course of cysticercosis, which is encountered with extreme rarity in man, is more or less latent, according to the locality attacked, the function of the organ involved and the number of migratory embryos, but it may give rise to very severe symptoms when it attacks the eye ground, the brain, or the spinal cord.

Fig. 47.

a *b*

Eggs of Tænia solium, magnified 470 times: *a*, under high power; *b*, under low power.

2. *Tænia saginata, mediocanellata* (the fat tapeworm) attains a length of eighteen to twenty-four feet, and even over. The head often more than 2 mm. thick, of cubic form, usually shows a marked brownish to blackish pigmentation, it has neither rostellum nor hooks, but has four powerfully developed suckers (Fig. 48). The sexually mature proglottides (Fig. 49) are 16 to 20 mm. long and 7 to 8 mm. broad. The uterus has on each side twenty to twenty-five delicate lateral branches, which branch dichotomously. Most of the proglottides, which are discharged singly, contain few eggs. These eggs differ from

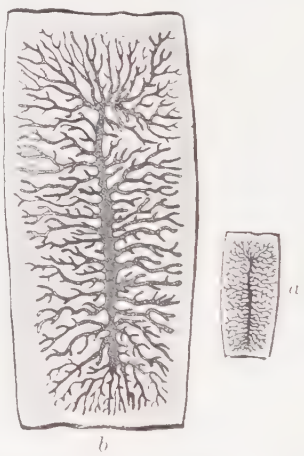
Fig. 48

a

b

Head of Tænia saginata: *a*, natural size; *b*, magnified eleven times.

Fig. 49.



a

b

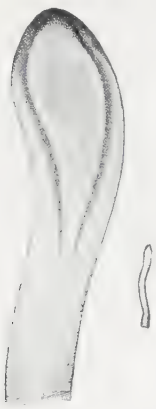
Single link of Tænia saginata: *a*, natural size; *b*, magnified four times.

those of the Tænia solium in their smaller size. This, however, is not constant. The statement that the eggs of the Tænia saginata have no hooklets is an error. The embryos of both tæniæ are armed. The infection of man with the Tænia saginata comes from eating measled meat. The cattle reinfect themselves through contact in the meadows with a species of Tænia saginata derived from man, or with their eggs, containing embryos.

3. *Bothriocephalus latus* (the broad worm) is named from the dimpled depression found at the sides of the wedge-shaped head (Fig. 50). It is the largest human parasite, attaining a length of thirty feet and over.

The ripe proglottides are only 5 to 6 mm. long and 12 to 15 mm. broad, and have in their centre a rosette-like marking, which represents the uterus packed with eggs (Fig. 51). Long sections of the proglottides are often passed with the stools in a macerated condition. The eggs are plentiful in the feces. They are .07 mm. long to .045 mm. broad, and yellow to brownish in color; and have a cap-like closure at one pole (Fig. 52). The escaping embryo, which has six hooks (called oncosphere), is decorated over the whole body with delicate cilia. It swims actively in water; and finally, either directly or through an intermediary host, enters certain varieties of fish (pike, perch, quab, and salmon),

Fig. 50.



Head of Bothriocephalus latus: *a*, natural size; *b*, magnified 17 times.

Fig. 51.

Links of Bothriocephalus latus, natural size.

Fig. 52.

Egg of Bothriocephalus latus, magnified 470 times.

in which it becomes encysted. By eating such fish or their products (such as caviare), man becomes infected, if the cysts have not been destroyed. Bothriocephalus disease is, therefore, to be found most frequently at the sea-shore and on the ocean. Of late years, however, the frequency with which such disease is encountered inland has increased considerably.

4. *Tænia cucumerina* (*elliptica*), the dog or cat tapeworm, becomes ten to thirty cm. long. The head has a powerful rostellum, with sixty hooks, arranged in four rows; and four suckers (Fig. 53). The ripe proglottides (Fig. 54) are 8 to 10 mm. long and 2 to 2½ mm. broad, and have a gray or reddish color—due to the shining through of the cocoon, which contains 6, 8, or 12 eggs (Fig. 55). The single egg measures .05 mm. in diameter, and contains an embryo with six hooks. These enbryos become encysted in dogs', as well as in human fleas—Pulex serraticeps and Pulex irritans and in dog's lice (trichodectes canis). Since dogs and cats fight their vermin by biting and pinching, or by licking their body, they swallow the intermediary host and infect themselves with the cysticercoids, which are again conveyed to children that are in the habit of playing with these animals; and it is a most striking fact that, up to the present time, the only case-reports of this disease

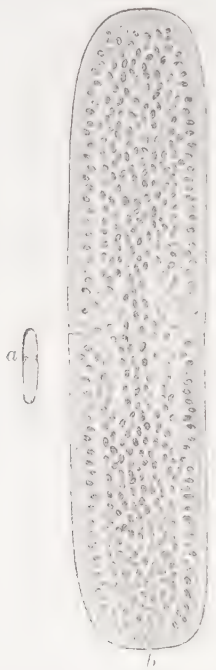
have concerned the presence of these parasites in children. From an observation of Köhl, about three weeks are required after the cysticercoid enters the body before ripe proglottides are passed. Asam-Huber have carefully studied the literature concerning these parasites.

5. *Tænia nana* (Fig. 56) has frequently been encountered in Italy, but only exceptionally in Germany. It becomes 2 to 3 cm. long, and half a millimetre thick. The rostellum has 25 hooklets and 4 suckers. On account of their small size, the proglottides passed do not usually help in the diagnosis. The eggs, round or oval, have a diameter of

Fig. 53.

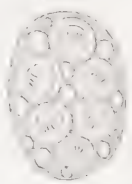
Head of Tænia cucumerina: *a*, natural size; *b*, magnified seventy times.

Fig. 54.



Link of Tænia cucumerina: *a*, natural size; *b*, magnified eight times.

Fig. 55.



Cocoon with eggs of Tænia cucumerina magnified 100 times. My own preparation.

.04 to .05 mm. The embryo has six hooks. The life-history of this tapeworm, which is widely distributed over Italy and Sicily, is not yet entirely known. According to Grassi, an intermediary host is not necessary to convey the parasite to man—a view that Leuckart opposes. The tæniæ nanæ infect children especially, and are found in enormous number—often 700 to 1000 in one individual. They have given rise to epileptiform attacks.

Symptoms.—What little we know of the symptomatology of tænia disease may be summed up as follows: In less than one per cent. of the individuals attacked, the Bothriocephalus latus gives rise to severe, and often fatal, anæmia. In other cases, like other tæniæ, it gives rise to no symptoms, or to no especial pathognomonic symptoms. Dyspeptic

disturbances, sour eructations, and nausea with vomiting—often headache and vertigo—are encountered. Colicky pains are frequent. Older children that know they have a tapeworm sometimes complain, just as is the habit of adults, of the crawling and biting of the worm. In tender and sensitive children, reflex symptoms, with epileptiform and choreiform conditions, have been observed. The previously mentioned formation of toxins by these parasites will explain the nervous symptoms, as well as the presence of anæmia, which is so frequent with tænia; but only those cases in which the whole symptom-complex disappears with the destruction of the worms can be considered positive; since it is evident that persons with epilepsy, chorea, and anæmia may become infected with tænia.

The **diagnosis** of the different forms of tænia is easily made, as a rule. The parents often bring links or portions of links passed, preserved in alcohol. In every case the true nature of these formations must be considered, and this may offer considerable difficulty. When the history is not clear after the careful passing of a rectal sound, the feces must be investigated for eggs; or a laxative may be given, which, as a rule, causes the evacuation of proglottides.

Fig. 56.

Tænia nana. Natural size.

The differential diagnosis between tænia solium and tænia saginata can usually be made by studying the links between two glass slides, but not always; since the individual proglottides may not show the branches of the uterus, on account of the absence of eggs (tænia saginata). The anamnesis, giving us a history of eating raw pork or beef, often leads to a correct diagnosis. Since the tænia solium must be removed as soon as possible, on account of the danger of cysticercosis, an exact diagnosis is of great importance—especially, since this disease gives greater chance for complete recovery after the removal of the worms than does that caused by the tænia saginata.

Treatment.—Since the usual remedies for tapeworm have a bad taste and are taken with difficulty; since they irritate the gastro-intestinal canal and sometimes cause severe toxic symptoms (especially the extract of filix mas), with lasting bodily injury, and even death; and since tapeworms do not, as a rule, give rise to alarming symptoms,—the institution of a cure for the condition is by no means a matter to be undertaken lightly and without careful consideration. A tapeworm cure must never be carried out without absolute certainty of diagnosis or without taking into consideration certain contraindications, such as early age; severe recent gastro-intestinal disease (appendicitis, peritonitis, typhoid fever, or other severe infectious diseases); heart disease with loss of compensation; severe phthisis; or recent recovery from abdominal operation. In such cases, one should hesitate at least for

a time, or should be satisfied with the throwing off of large sections of proglottides; in order to avoid the danger of cysticercosis.

The day before the administration of the cure, the intestine must be thoroughly evacuated by means of castor-oil, Hunyadi water, etc.; and the children must receive a bland diet. The following day, tea or coffee must be given for breakfast; and an hour later, the vermifuge. In the Kaiser Francis Joseph Children's Hospital at Prague, in Professor Ganghofner's clinic, the tapeworm remedy prepared by H. A. Jungclaussen, of Hamburg, has been used exclusively in later years. This is called Cucumerin, and is a concentrated extract of 300 grams (9 oz.) of pumpkin-seeds. It has been extensively used in Mexico as a tapeworm remedy, and has been warmly recommended by v. Storch of Copenhagen. The bottles contain about 40 grams (1 oz.). It tastes like beef-juice, and can be given dissolved in soup or cocoa, one or two hours after breakfast. It is nearly always well taken by the children. Two hours later, a purge of castor-oil, for example, may be given; a tablespoonful every half-hour, until free stools result. Large intestinal irrigations are also of use. The results are not always equally good, and it is not easy to say whereon this depends. Sometimes repeating the cure once or twice produces the desired result.

After each tapeworm cure, the children must be nourished mainly on soup and broths for two or three days. The worms and links that have been passed should be burned, and not thrown into the closet or sink; and any vessels that have been used to receive the evacuations should be carefully disinfected with boiling water and cleansed. In the interests of all those that come into contact with the patient, the strictest cleanliness must be observed.

In former days, the favorite remedy was ethereal extract of filix mas, in a dose of 0.5 Gm. (7 gr.) for each year of the child's life; but never exceeding a maximun dose of 5. Gm. (70 gr.). We also used for one year the flowers of kousso; and of these we gave 1.0 Gm. (15 gr.) for each year of the child's age, but never exceeded 8.0 Gm. (120 gr.) in older children. These remedies may be given floating on tea or syrup, and the children may be persuaded to drink them quickly by promising them candy or some other dainty afterward. After taking the vermifuge, a rest in bed was ordered in all cases, to avoid the marked tendency to vomiting that often develops. At the end of two or three hours, the patient received a teaspoonful of the freshly-prepared vermifuge every fifteen or thirty minutes, until the effect was produced; and in addition, often a rectal enema.

The dose of canella, which may be given in tablets or pills, or mixed with sugar, is 3–5–8 Gm. (45, 75, 120 gr.). As to pomegranate bark, (which can be taken as a decoction of from 5 Gm. (75 gr.) to three-fourths of an ounce of the bark, macerated in 200 c.c. (six ounces) of water for

twenty-four hours, of which three to four portions are taken in the course of a day), pelletierin, and koussin, I have no individual experience. The tapeworm tritol (Dietrich) (a jelly-like emulsion of extract of filix mas, diastasic malt, and castor-oil), as well as filmaron and teniol, I have not personally used.

Active **prophylaxis** should consist in a knowledge of the life-conditions and the mode of introduction into the organism of the different forms of tænia. The more generally such knowledge is popularly distributed, the sooner may we hope that certain forms of tænia will become very rare among educated persons, or perhaps disappear entirely from view.

IV. THE ECHINOCOCCUS

The Tænia echinococcus (Fig. 57) is a broad tapeworm, .2 or .3 mm. wide; and reaching a length of, at most, 5 mm. It is often found in enormous numbers in the intestine of the dog and other animals, such as wolves, jackals, and foxes. Microscopically, the head is armed with a claw-like rostellum and four suckers. Of the three progrottides, the last one contains from four hundred to five hundred eggs, which have a diameter of .02 mm., and contain an embryo with hooklets.

Fig. 57.

Tænia echinococcus, natural size.

When the eggs reach the gastro-intestinal canal of man or other omnivorous or herbivorous animals, the embryo becomes free; and, partly actively and partly passively, through the blood and the lymph-stream, reaches the different organs of the body—and most frequently, the liver.

There develops gradually from the embryo a cyst filled with fluid, the acephalocyst. Slowly this becomes larger. A cuticle several layers thick develops; also an intercellular parenchymatous layer. The organ affected produces itself a connective-tissue capsule around the cyst (Fig. 58). Small capsules are formed within the cyst, by the proliferation of the parenchymatous layer, from whose inner wall pedunculated heads spring, having a circle of hooks and four suckers. The whole picture represents the measled stage of the above-described echinococcus tapeworm, and is called for brevity echinococcus.

1. In the above-described form, we have the *Echinococcus simplex.* Then similarly constructed formations develop from the wall of the mother-cyst (acephalocyst), which are called daughter-cysts. On the parenchymatous layer of these, we find the formation of breeding-chambers with heads. We may find the formation of granddaughter-cysts from the daughter-cysts.

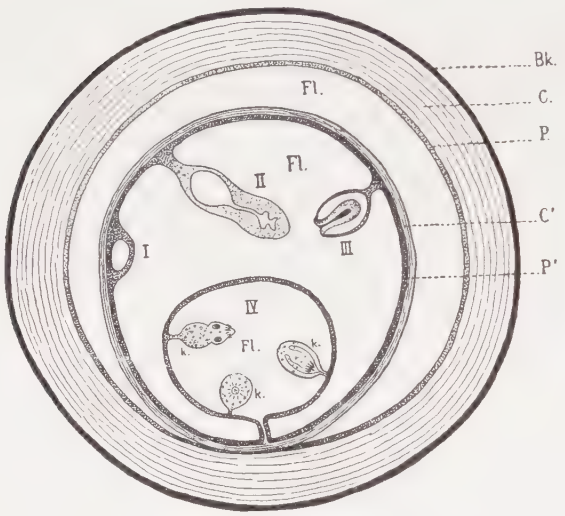
2. This form is called the *Echinococcus compositus hydatidosus* (E. compositus endogenus). It is the most frequent form of echinococcus found in man. Since the proliferation of the parenchymatous layer

occurs within the cyst, these two forms are also designated echinococcus endogenus (Fig. 58); but when the proliferation of the parenchymatous layer breaks through the cuticular layer externally, and the daughter or granddaughter-cysts become situated outside the mother cuticle, then there results:

3. *The Echinococcus ectogenus,* or granulosus, which is found in man in the omentum, the peritoneum, etc. (Fig. 59).

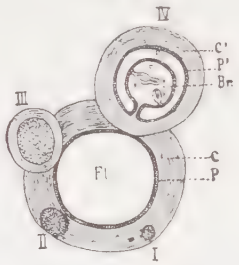
4. A sub-variety of the latter is the *Echinococcus multilocularis,* which represents a group of very small, closely packed cysts, and may reach the size of a human fist. In sections of the same (Fig. 60) one sees nothing but small cavities with jelly-like contents, containing the heads and scolices. The echinococcus multilocularis seems to arise from continued budding, and is often found in the liver of man.

Fig. 58.



Echinococcus compositus hydatitosus (endogenus). Bk, the connective-tissue capsule. C, the cuticular layer of the mother-cell, or cyst. P', the parenchymatous layer of the mother-cell. C', the cuticular layer of tne daughter-cell. P', the parenchymatous layer of the daughter-cell. Fl, the fluid contents. I, II, III, IV, stages of development of the head of the echinococcus (k) and the breeding-capsule (IV).

Fig. 59.



Echinococcus ectogenus. C, the cuticular layer of the mother-cyst. P, the parenchymatous layer of the mother-cyst. C', the cuticle of the daughter-cyst. P', the parenchymatous layer of the daughter-cyst. k, the echinococcus head. Br, the breeding-chamber.

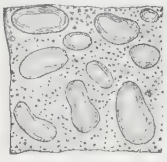
All four varieties of echinococcus are derived from the tænia echinococcus of the dog.

Within the echinococcus bladder, we find a clear, slightly opalescent fluid, with a specific gravity of 1.009 to 1.015. This contains traces of albumin, sugar, inosit, leucin, tyrosin, cholesterin, hematoidin, and succinic acid, together with the sodium and calcium salts of the latter. Out of 1.5 per cent. of inorganic substance, .5 to .8 per cent. are sodium chloride. Brieger found present a substance with poisonous properties.

Symptoms.—In regard to the symptomatology of the echinococcus in man, I can speak only from a general standpoint. Since the echinococcus is found sometimes singly and sometimes with several species

in one or several organs of the body, its action may be manifold. This is evident from its size and the conditions of its life, as well as from the effect on the function of the organs affected. The embryo, which wanders in, sets up first local irritation, which the organ affected meets by enclosing it in connective tissue. This encapsulation does not occur in cavities. The growth of the acephalocyst is very slow for several months. The increase in size causes, at first, no difficulty for the host. It is painless, but when a certain size has been reached pressure symptoms arise. After further growth, these symptoms are not confined to the organ affected, but extend to the neighboring parts of the body. The echinococci enter the adjoining parts of the body either spontaneously or as the result of trauma. They frequently enter the cavities of the body; and the resulting absorption of echinococcus-fluid results in repeated attacks of hives and severe, even fatal, intoxication. The parasite can exist in some organs for years, without affecting its host. On the other hand, it may unsuspectedly lead to sudden death, or set up a chronic marasmus. The echinococcus may die, and become encapsulated; it may also spread, and lead to fatal pyæmia. The case-reports on echinococcus disease are unusually plentiful.

Fig. 60.



Section through a portion of Echinococcus multilocularis, magnified five times.

Man usually acquires the echinococcus from the dog, which is so frequently the intermediary of the tapeworm. Echinococcus-cysts are often given to the dog in meat at slaughter-houses, butcher-shops, and provision-stores; and even in private houses. The dog that carries the tænia echinococcus may directly infect man with the eggs, by licking him; or they may be deposited by the dog on various articles of use, such as plates, drinking-vessels, etc., and so indirectly reach human beings. Dogs that are free from echinococcus may also become the carriers of the eggs through sniffing and smelling infected animals or their evacuations. Considering these facts, the physician should hold it his duty to warn parents of the danger that can arise from intimate contact with dogs.

Fig. 61.

Echinococcus-hooklets, enlarged two hundred times. My own preparation.

The **diagnosis** of echinococcus is made probable by the discovery of a cystic tumor, commonly located in the liver, especially if close contact with a dog has occurred. The hydatid thrill, which may sometimes be demonstrated by percussion and palpation, depends upon the trembling of the hard, gelatinous mass, which is made to vibrate; and this thrill is a decided help in diagnosis. The diagnosis is positive only when the hooklets (Fig. 61), the heads of the parasite, or portions of the cuticular layer are found in the fluid evacuated spontaneously after puncture or incision. Chemical investigation of this fluid will give further diagnostic aid, as will

also the formation of precipitin in the same when brought into contact with the serum of a tænia immune (Langer, 1905). The leakage of echinococcus-fluid following exploratory puncture, with all its harmful results, must be kept in mind in every case.

The **prognosis** depends upon the size of the echinococcus, the function of the organ affected, and the possibility of operative extirpation—which is, after all, the only rational therapeutics.

Genuine **prophylaxis** comes from our knowledge of the life-conditions and the mode of transference of the parasite to man. Anthelmintic cures should be carried out, from time to time, on dogs that are house-companions; and the possibility of fresh infection should be guarded against as much as possible.

DISEASES OF THE PERITONEUM

BY
PROFESSOR M. STOOSS, OF BERNE

TRANSLATED BY
DR. CHARLES F. JUDSON, PHILADELPHIA, PA.

I. ACUTE PERITONITIS

ACUTE inflammations of the peritoneum most frequently arise by direct extension from a neighboring organ; less often, the peritoneum is infected by way of the circulation—hæmatogenous peritonitis. The source of infection may be the gastro-intestinal canal, the liver and the gall bladder, the kidney and the bladder, and also the female genitalia. Inflammatory processes in the peritoneum may proceed from the retroperitoneal lymph-nodes and the lymph-nodes of the mesentery.

In infancy, acute peritonitis is most frequently the result of inflammation of the appendix. Exceptionally, intestinal ulcers are the cause; while perforation of typhoid ulcers occurs much less often than in adult life. Since round ulcers of the stomach and malignant new growths are extremely rare, we seldom, if ever, encounter peritonitis dependent upon perforation of the stomach. The liver and gall bladder rarely give rise to peritonitis, since gall-stones, as well as abscesses of the liver, are uncommon in childhood. Echinococcus cysts are even more rare, and are only exceptionally encountered in our section of the country. The urinary tract scarcely requires consideration; on the other hand, the female genital organs may be a portal of entry. We do not meet the septic inflammations that are so common in adult life.

The most important *micro-organisms* in the causation of the disease are the bacterium coli commune, the streptococcus, the pneumococcus, the staphylococcus aureus, and the gonococcus. Single or multiple infection may occur; when the disease is due to perforation of the gastro-intestinal tract, many varieties of aërobic and anaërobic intestinal bacteria invade the peritoneal cavity.

In considering this subject, we shall endeavor to set forth those features that are peculiar to infancy and are most often encountered at this time of life. Perforation-peritonitis runs its course as in adults, and need not be considered; also inflammation after intestinal occlusion and with incarcerated hernia. Traumatic peritonitis requires no special mention. Septic inflammations are considered with peritonitis in

infants, and the newborn. Inflammatory processes originating in disease of the appendix are considered in a special section of this work.

On the other hand, some forms of peritonitis are peculiar to childhood. It is the common experience that pneumococcus and streptococcus infections are more easily acquired in childhood. Examples may be cited; such as pneumococcus and streptococcus osteomyelitis, pneumococcus otitis, and pneumococcus empyema, which occur so seldom in later life. Both pneumococcus and streptococcus peritonitis form well-defined pictures in early life, They will, therefore, receive special consideration and serve as the type of acute peritonitis. Inflammations of the peritoneum resulting from vulvovaginitis are of special interest to the pediatrist; and they, as well as peritonitis in the newborn and in infants, will receive mention.

1. PNEUMOCOCCUS PERITONITIS

In recent years, much has been written, especially by French physicians (Netter, Brun, Broca, Comby), describing the type of peritonitis in which the pneumococcus of Fränkel is regularly found as the sole etiological factor. The clinical picture is no new one. Already, in 1842, Du Parque described similar cases under the title of "essential peritonitis of young girls." Rilliet and Barthez mention them, as does West; and Gauderon collected in his thesis twenty-five cases (1876). In Gerhardt's Handbuch, Rehn discussed "idiopathic rheumatic peritonitis." In former years, Henoch observed suppurative peritonitis.

Symptoms.—According to my observations, the disease runs the following course: The onset is sudden, with very acute symptoms, and apparently without cause. The exudation of pus is considerable, sometimes several quarts; and it tends, in the majority of cases, to collect in the lower part of the abdomen, where it is separated from the intestinal coils and encapsulated. Left to itself, the exudate is apt to penetrate the abdominal wall through the unbilicus. In a lesser number of cases, encapsulation does not occur; and we have diffuse suppurative peritonitis.

The patients, usually girls in the middle period of childhood, are taken sick with colicky pains, commonly located in the lower part of the abdomen, with vomiting and high fever, with which severe diarrhœa is usually associated. After a few days, the extremely violent initial symptoms moderate. The pains become less, vomiting ceases, and the fever is less high. The loose stools, however, persist. Soon after the beginning of the disease, the abdomen becomes markedly distended (meteorism).

The next stage of the disease is characterized by a collection of fluid in the abdomen, and sets in after about fourteen days. Empyema of the abdominal cavity is present. The general condition may now appear somewhat improved.

In the case of an eleven and a half year old girl, who walked to the clinic, the pus was visible through the umbilicus, and on evacuation, next day, amounted to one and a half quarts.

On palpation, we find a tense, elastic abdomen, and often evident fluctuation. Percussion reveals dulness, which may reach above the umbilicus. The outline of dulness in my cases was represented by a curved line, with the concavity upwards; in one case only the dulness assumed the form of an ovarian tumor, with marked prominence below the navel. If the abscess is not opened at this stage of the disease, the patients lose flesh rapidly, and have continued fever; while the circumference of the abdomen constantly increases. The umbilicus protrudes, from the pressure of the exudate, like a hernia; the skin becomes tense; and the exudate may show through it. Spontaneous perforation through the abdominal wall may follow.*

When the exudate is not rapidly walled off from the intestinal convolutions, diffuse suppurative peritonitis sets in, which naturally gives a much more serious prognosis; but even this form may end in recovery, as the following case, which came under my observation, shows:

A twelve-year-old girl, who had been delirious for some days, was brought into the hospital on the eighth day of her illness. The tongue was dry and brown, the eyes hollow and circled by dark shadows. At the operation, nearly two quarts of thick, greenish yellow pus were evacuated. There were no adhesions. Immediate improvement in the general condition resulted, and complete recovery followed after several weeks.

The **diagnosis** is difficult in the early stages. The condition is usually mistaken for appenditicis and typhoid fever; or, in the later stages, for tuberculous peritonitis. Important for the *differential diagnosis* are the following: In appendicitis, constipation is the rule, diarrhœa being decidedly exceptional. Tension of the abdominal wall on the diseased side is nearly always present in appendicitis; whereas in pneumococcus peritonitis it is absent or scarcely noticeable. In the early stages, the disease may easily be confounded with typhoid fever. Such was the case with one of my patients. Severe and widespread pains in the abdomen, with protracted vomiting, speak against typhoid. The Widal reaction and the leucocyte count will help in the diagnosis. Should doubt persist, one must carefully observe whether an exudate is present in the abdominal cavity. If the patient is first observed after pus has collected, tuberculous peritonitis may be suspected. Of great importance for our decision is the mode of onset. Tuberculous peritonitis rarely begins acutely, and the formation of an exudate pushing forward the umbilicus is unlikely within such a short time. Those that

* Hagenbach-Burckhardt observed a case in which the pus made its way to the thigh.

are familiar with pneumococcus peritonitis will probably be able to make a diagnosis when an exudate is present, as I succeeded in doing on two occasions.

At the operation, we usually encounter a single large sac, completely walled off from the intestines; usually lying in the middle of the lower half of the abdomen—less often, laterally; and containing large quantities of thick, creamy, greenish, yellowish green, or yellow, odorless pus, containing many flakes of fibrin. The appearance resembles thoracic empyema. The rapidly fatal cases present the picture of pneumococcus sepsis; in those which run a less rapid course, the purulent exudate is diffused throughout the abdomen and there are few, or no, adhesions.

How do the pneumococci reach the peritoneal cavity? In a certain proportion of cases, infection must occur by way of the circulation. Michaut, in fact, considers this to be the only path of infection. Considerable evidence is extant that the cocci reach the peritoneum through the intestines or appendix (de Quervain). The frequent occurrence of the disease in girls naturally points to the genital tract as the starting-point in some of the cases; whereas in others it is probable that the infective agent passes directly from the pleural to the peritoneal cavity through the diaphragm.

To illustrate the great variety of conditions encountered, we may cite the fact that one of our patients, on the third day of the disease, showed, in addition to the symptoms of peritonitis, a pneumonic infiltration of the left upper lobe. In another, on the first day of the disease, tonsillar angina and pleuritic friction were present. In a third case nephritis and pleurisy preceded the peritonitis. The conception of an "idiopathic" peritonitis can no longer be maintained.

Treatment must be medical at first. As soon as the tentative diagnosis of acute peritonitis is made, absolute rest must be enjoined, especially complete rest of the intestinal tract. It is of special importance in pneumococcus peritonitis, in which there already exists a tendency to wall off the exudate, to favor this process and prevent the inflammation from becoming general.

During the period of acute symptoms, the administration of food by the mouth must be absolutely forbidden, and the patients nourished by nutritive enemata and rectal injections of water. I hold the identical views regarding the absolute importance of this measure that Sahli has expressed. Should the rectal injections not be retained, on account of irritability of the intestines, subcutaneous infusions of normal salt solution must be given, and are of great value.

Rest for the intestinal tract is secured by the use of opium (the only drug of any service), in doses ranging from one to ten drops of the tincture opii, according to the age of the child; or from one-twelfth to

one-fourth of a grain of the extractum opii, repeated several times, until the pain ceases, when the drug should be withheld. Should the pain return, the cautious continuation of the treatment with opium in small doses is necessary.

Locally, we use the ice bag (partly filled to avoid pressure on the abdomen) or iced compresses, whereby, as a rule, much relief is afforded. Should this not be the case, lukewarm compresses may supply their place and prove grateful to the patient. The influence of local treatment on the course of the disease is not definitely known. Leeches frequently afford much relief from pain, but their use may be dispensed with unless the suffering is extreme. Purgatives are strictly interdicted, as well as intestinal irrigation; at least, in the early stages of the disease. As soon as an exudate develops, but no sooner, laparotomy must be performed.

The incision should be central, two to three inches long, below the navel. Drainage must be used. In the Jenner Hospital, we irrigate the abdominal cavity with normal salt-solution or Tavel's solution ($7\frac{1}{2}$ per cent. sodium chloride and $2\frac{1}{2}$ per cent. calcined sodium carbonate); others omit irrigation. The results are excellent in the encapsulated form of the disease; in the general peritonitis, the results are better, the earlier the operation is performed. Without operation, the patient is exposed to the dangers of septicæmia. Should spontaneous rupture through the abdominal wall occur, the conditions are rarely favorable for drainage.

2. STREPTOCOCCUS PERITONITIS

Streptococci may apparently cause primary inflammation of the peritoneum. Such cases occur less often than pneumococcus peritonitis, but are more dangerous. They have this in common with pneumococcus peritonitis; that they set in very acutely and apparently without cause, and are usually accompanied by very violent diarrhœa. Vomiting, fever, and pain are likewise present; but the tendency of the disease noticed in the former group of cases to become circumscribed is rarely observed, and most of the numerous cases reported end fatally in a few days. The purulent exudate is generally thin and reddish yellow.

The case that follows illustrates the course of this disease: A six-year-old girl was taken sick subsequently to a light attack of jaundice, with high fever and violent abdominal pain, accompanied by vomiting, and distention; and, soon after this, with loose, offensive stools. On the fifth day after admission to the hospital, almost three-fourths of a quart of pus was evacuated at the operation, giving a pure culture of streptococci. Three days after this, death occurred. Besides the lesions of the peritoneum the autopsy revealed merely a hyperæmia of the mucous membrane of the stomach and of the ileum, with small

hæmorrhagic areas. The appendix was intact. Figure 62 portrays an encapsulated peritoneal abscess in the left hypochondrium, in a five-year-old girl, which began with the same violent symptoms, some weeks before her entrance into the clinic, where she recovered after operation. The bacteriological findings were streptococci and colon bacilli. In this case, the process had become circumscribed—a rare occurrence, possibly due to the presence of colon bacilli.

Fig. 62.

Encapsulated streptococci—coli peritonitis. Girl five years old. The form of the abdomen is different from that seen in tuberculous peritonitis. Compare Fig. 63, page 262.

In this category belong those cases of peritonitis that exceptionally follow in the wake of the infective fevers; such as scarlet fever, diphtheria, measles, erysipelas, and acute tonsillitis. Henoch has frequently seen postscarlatinal peritonitis, especially when nephritis was present; and Heubner designates as a peculiarity of streptococcus sepsis in scarlet fever, the tendency to suppurative inflammation of the serous membranes.

The **treatment** of diffuse streptococcus peritonitis is most unsatisfactory. Considering the fact that the chances of recovery are slight, surgical intervention should be made at the earliest possible moment. In any event, the mortality in these cases is very high. Polyvalent streptococcus serum, as prepared by Tavel, Van de Velde, and Aronson, should be injected as early as possible. Intravenous collargol injections may also be given.

3. GONOCOCCUS PERITONITIS FOLLOWING VULVOVAGINITIS

Vulvovaginitis may lead to peritonitis, through extension upward of the disease. The complication is, however, comparatively rare in proportion to the very numerous blennorrhœal diseases of the vulva and vagina in young girls. Nevertheless, the contributions to the literature of this subject within recent years (Marfan, Comby, Baginsky, et al.) prove that gonococcus peritonitis is no great rarity.

Experience teaches that the intensity of the inflammation varies considerably. Besides very mild cases, we encounter very severe manifestations of the disease.

The mild cases described by Marfan and Comby also begin very acutely, with vomiting, fever, and severe abdominal pains; but these subside within a few days. This is not an infrequent form of the disease, and deserves careful attention. When the process becomes more extensive, with the signs of acute general peritonitis, the sensation of pain is localized chiefly in the lower part of the abdomen; so that appendicitis may be simulated and operation performed. Even the cases that present very severe symptoms—enormous abdominal distention, very high fever, rapid and irregular pulse, dry tongue, and extreme weakness—may recover without operative intervention. Zaradorsky reported seven cases, two of them with bacteriological and post-mortem findings. Two other cases, in which the patients died after operation, come from the Johns Hopkins Hospital. I have observed the following case:

An infant a few weeks old, in miserable condition, was admitted to the Jenner Hospital on account of severe diarrhœa. The profuse yellowish green discharge from the genitalia contained many gonococci. The abdomen was meteoristic, but not tense. The eyes showed conjunctivitis and keratitis. The child became steadily worse, and died suddenly. At the autopsy, twenty to thirty centimetres of a cloudy, yellowish fluid, with numerous fibrin-flakes, were found in the lower pelvis. The peritoneal covering of the small intestines, as well as that of the stomach, spleen, and liver, showed, in a few places, fibrinous exudate. On pressure in Douglas's cul-de-sac, a considerable quantity of fibrinopurulent fluid was evacuated. The uterine cavity contained a little thin pus; the mucous membrane was hyperæmic; there were no changes in the tubes and ovaries; the vaginal mucous membrane showed hyperæmia, with scattered hæmorrhages; in Douglas's pouch there were many fibrin-plaques; the intestinal canal showed no acute inflammatory changes; the mucous membrane in the upper part of the small intestine was evidently thickened. Anatomical diagnosis, gonorrhœal vaginitis and endometritis; diffuse, fibrinous, suppurative peritonitis; chronic enteritis. Gonococci were present in the peritoneal exudate.

Gonococcus peritonitis, accordingly, may limit itself to the neighborhood of the genitalia, or may become general. In the latter cases, one finds, either at laparotomy or at autopsy, more or less thin pus in Douglas's pouch, besides diffuse fibrinopurulent deposits. The knowledge we have acquired of these forms of peritonitis makes a careful investigation of the genital organs of young girls imperative, even when the signs of peritonitis are not marked.

Gonococcus peritonitis can easily be confused with appendicitis. Important for the differential *diagnosis* are the absence of pain at McBurney's point, the absence of muscular rigidity, and the presence of a vaginal discharge.

Treatment must be primarily medical, according to the principles laid down for cases of acute peritonitis. Operative intervention should be called for only in the desperate cases; for we know that even severe cases can get well without it. Nevertheless, the question of operation must be carefully considered in every case, in spite of the comparative benignity of gonococcus peritonitis. One should not wait until the patient's condition has become hopeless.

PERITONITIS IN THE NEWBORN AND IN INFANTS

Peritoneal inflammation may arise in fœtal life, and lead to death of the fœtus in utero or to malformations of the intestines and bile-ducts (atresia). The cause may be syphilis, or not determinable.

In former years, peritoneal infection in the newborn, beginning in the umbilical wound, occurred epidemically in lying-in hospitals, together with puerperal fever; so that Bednar could assert that peritonitis was more often encountered in the newborn than at any other period in the child's life. Of late, this form of peritonitis is seldom encountered; just as severe infections through the umbilicus have become rare.

Arteritis, gangrene of the umbilicus, and erysipelas are the most common causative factors.

Arteritis was the starting-point of acute general peritonitis in the only case I have observed in recent years. Suppuration had extended to the tunica vaginalis, and had led to abscess-formation (staphylococci) on the posterior surface of the testicle.

The **diagnosis** of peritonitis in infancy is often peculiarly difficult; many cases certainly are overlooked. Abdominal distention, with meteorism and colicky pains, is so much an every-day occurrence in infants that one may easily fail to recognize those cases of peritonitis in which the amount of the inflammatory exudate is not large. There may be no vomiting, and little or no fever. The highest temperature recorded in the case of gonococcus peritonitis reported above was 37.8 C. (100° F.). On careful examination, we shall usually recognize general peritonitis by the marked tension of the abdomen and the exquisite sensitiveness on pressure. The latter symptom may also be absent, as well as fever and vomiting.

The diagnosis is easier when the disease begins suddenly in fairly healthy children.

An infant four weeks old, whom I was treating for dyspepsia, suddenly began to suffer severe abdominal pain with abdominal distention and constipation. The child cried constantly; the abdomen was hard and tense, but there was no fever or vomiting. Death followed on the third day. Postmortem showed a diffuse fibrinous and suppurative peritonitis, but no free exudate. The navel and the umbili-

cal vessels were perfectly normal; there was a mild grade of intestinal catarrh, but no ulceration. The appendix was normal. The bacteriological investigation showed only the bacterium coli communis.

In infancy, the **etiology** of peritonitis is somewhat different from that in older children. Appendicitis, the most frequent cause in children over two years old, is rare in the first year of life; as is also encapsulated pneumococcus peritonitis. From a study of my autopsies, I should consider peritonitis in the first two years of life as a manifestation of that symptom-complex which Heubner has described in his text book as "multiple suppurative inflammation of the serous membrane." In accordance with Heubner's description, we find, associated with suppurative pericarditis and suppurative peritonitis, pleurisy with a thick, fibrinopurulent, circumscribed exudate, without fluid pus, whence the infection seems to have spread. In one of my cases, meningitis was also present. Pneumococci and streptococci are the exciting factors.

In the second class must be considered those cases of peritonitis which originate in the intestines. It is a fact generally known that peritonitis is a quite unusual complication of the catarrhal diseases of infancy. I must, however, admit, after looking over my post-mortem records, that I have seen peritonitis associated with every form of severe enteritis; and even with the milder catarrhal affections, as the foregoing case of colon bacillus peritonitis shows. Baginsky had the same experience. Heubner emphasizes the occurrence of peritonitis in enterocolitis. Most of the patients that I have seen were but a few weeks old.

I have seen perforation peritonitis in infants follow ulcerative processes, especially in the duodenum. In the third place, we must recollect that vulvovaginitis is by no means rare, even in the earliest period of life. The above-mentioned case of gonococcus peritonitis occurred in a child only a few weeks old.

Peritonitis as a symptom of general sepsis is by no means so frequently encountered in the newborn and the infant as earlier writers have reported; on the contrary, the peritoneum is usually intact, and one sees merely marked injection or small hæmorrhages (Fischl).

II. TUBERCULOUS PERITONITIS

Tuberculosis of the abdominal peritoneum, with a few exceptions, runs a chronic course. However, we have recently been reminded that tuberculous peritonitis may begin under the guise of acute perityphlitis (appendicitis). Such cases have also been described in children.

The view has long been held that the great majority of chronic inflammations of the peritoneum are of tuberculous nature. As early as 1850, Aran declared in his clinic that one would find tubercles in the peritoneal cavity in most cases of chronic peritonitis. It was not until the tubercle bacillus was discovered and until after König, in 1884, had

introduced laparotomy in the treatment of tuberculous peritonitis, that it became possible to determine definitely the etiology and course of this disease, as well as to differentiate it from other diseases of the peritoneum. Even to-day, we have not reached complete accord; especially concerning the existence of a nontuberculous chronic serous peritonitis.

Tuberculosis of the peritoneum occurs comparatively often in childhood, and deserves special mention from the *pathological standpoint.*

Laparotomies on patients that have suffered for only a comparatively short time with serous exudation in the abdominal cavity have enabled us to study the early stage of tubercle development in the peritoneum. In these cases, we find the same crop of minute tubercle-nodes that we find occasionally in acute miliary tuberculosis; the serous coat shows almost no change. The tuberculous process may remain stationary or may completely retrograde, with disappearance of the tubercles, leaving the serosa uniformly smooth and shining.

In a more advanced stage, conditions are essentially altered. The tubercles become larger and caseated centrally, and the serous coat is thickened as the result of the reactive inflammation. The ordinary anatomical form of tuberculous peritonitis gradually develops, with pseudomembranous formation and with adhesions between the abdominal viscera and the peritoneum. The plastic fibrinous exudation leads to adhesions, and the tubercles become confluent and form larger cheesy nodes. Around these caseous foci, we always find a crop of gray miliary tubercles, partly caseated centrally. The omentum usually shows marked evidences of tuberculous disease. The tubercles become confluent, forming very large nodes. Adhesions are formed with the coat of the intestines and of the abdominal wall, giving rise to the well-known tumor-like band, which runs transversely or directly across the navel (gateau abdominal).

The exudate may never be large, but seldom fails completely. Out of forty-two cases, Borchgrevink found not a single one without some exudation. Strictly speaking, dry, fibrinous peritonitis must be exceptional. The exudate is usually clear and thin; at times, greenish and turbid, from the admixture of fibrin-flakes.

Hæmorrhagic peritonitis is rare, especially in childhood. Bacilli are not plentiful in the exudate. Encapsulation usually results after the disease has existed for some time, with the formation of intestinal adhesions. Even in this stage, the process may undergo retrograde changes. The granulation-tissue undergoes fibrous transformation; cheesy deposits that are not absorbed may be encapsulated in the connective tissue. We then find peritoneal thickening and adhesions as the sole residue of the tuberculous process, which has run its course. The very marked tendency of peritoneal tuberculosis to get well has been especially emphasized by the investigations of Borchgrevink.

Infection of the exudate, especially from the intestines, may give rise to suppurative, walled-off abscesses, which may make their way through the abdominal wall (especially the umbilicus) or may involve the contiguous organs. Exceptionally, they may heal without perforation or operation. In the abdominal organs, we see a diffuse crop of gray or cheesy tubercles. The intestines are rarely affected; exceptionally, fistulas follow perforation of a suppurative focus in the intestines. In childhood, tuberculous ulcers of the intestines, which might be considered the starting-point of tuberculous peritonitis, are decidedly rare. Perforation of the bladder-wall rarely occurs. An extensive cheesy tuberculosis of the tubes, and also of the body of the uterus is by no means rare in girls, analogous to the conditions in the adult.

Symptomatology.—The French authors have differentiated several varieties of this disease. From the pathological and clinical standpoint, I shall attempt to differentiate only the common types of the disease and that known as chronic serous peritonitis. Chronic ascites is very frequently the earliest and the most important sign of tuberculosis of the peritoneum in childhood, and gives us a well-defined type of the disease.

CHRONIC TUBERCULOUS ASCITES

This disease often attacks children that are apparently healthy. An insidious onset is by no means the rule. More frequently, vague abdominal pains, with moderate fever, nausea, and vomiting, usher in the disease. Marfan has drawn our attention to the frequency of pleuritic friction at the base of the lungs, associated sometimes with exudation into the pleura. These symptoms soon vanish, and a considerable free exudate accumulates in the abdomen, which may be the only sign of the disease for weeks or even months. Since the initial symptoms last only a short time, and are not very severe, the physician usually first observes the cases after the marked distention of the abdomen has attracted the attention of the child's caretakers, when the exudate has reached considerable dimensions.

The general health is not seriously affected. Wasting, which is so marked in the later stages of the disease, in contrast to the distended abdomen, is slight or absent; and the investigation of the other organs shows no signs of tuberculous disease. The exudate may remain stationary for months together, or at times disappear, only to reappear again. When it is on the increase, nausea and vomiting, with slight febrile disturbances, show the reaction of the organism. The evacuations are at times somewhat loose and diarrhœal; sometimes clay-colored. Again, they may be constipated. The appetite is variable. The exudation may entirely and permanently disappear, as well as all signs of illness; and the health be completely restored. In other cases, tuberculosis develops subsequently in some other part of the body.

I found, in a child that had been operated upon for intestinal occlusion due to a traumatic hæmatoma of the intestinal wall, unsuspected tuberculous nodes on the peritoneum. When I saw this patient again, many years later, I learned that there had been no further symptoms of abdominal tuberculosis. On the other hand, she then suffered with tuberculous spondylitis.

The **physical signs** are those of every free fluid exudate in the abdomen. The exudate may be so large that the abdominal wall becomes tense and the navel protrudes. The demonstration of any considerable amount of ascites offers no difficulties, but it may be more difficult to determine the primary causal factor in the disease. Besides tuberculous peritonitis, we must consider as possible causes of the ascites, general circulatory disturbances due to heart disease, Bright's disease, cirrhosis of the liver, abdominal tumors, general cachexia, and chronic serous peritonitis.

FIG. 63.

Chronic tuberculous ascites. Boy four years of age.

Ascites of cardiac origin is associated with other signs of passive congestion; also ascites due to nephritis. The differentiation from atrophic cirrhosis of the liver may, now and then, offer a difficulty; but the cirrhoses are comparatively seldom encountered in childhood, and the diminution in the size of the liver can be demonstrated objectively after abdominal puncture. In doubtful cases, the investigation of the exudate may aid the diagnosis. A large number of lymphocytes always speaks in favor of a tuberculous process.

Abdominal tumors must be considered in making our diagnosis, when nodular tumors are present in addition to the exudate. The mode of development of the disease and the consideration of the symptoms as a whole will help us to decide. In addition, the character of the exudate must be determined by bacteriological and cytological examination.

Chronic Exudative (Nontuberculous) Peritonitis is certainly a very rare disease. Not a single case has been confirmed by autopsy. Still, we cannot deny that there occur at the time of puberty cases that have

only a small abdominal exudate and can hardly be considered tuberculous (Quincke). Possibly they depend on functional disturbances of the heart and alterations in the composition of the blood. Exploratory puncture, injections of tuberculin, and inoculation experiments in the laboratory will throw light on the case. In general, the practitioner should consider all cases of chronic peritonitis with exudate for which no definite cause can be found as most probably tuberculous.

Caseous peritonitis may develop from the exudative type, if the organism has not the power to overcome the disease in the beginning. More exceptionally, there is no demonstrable fluid in the peritoneal cavity at any time in the disease. We can no longer demonstrate the presence of movable fluid; and the areas of percussion dulness are little, if any, changed by alterations in the position of the patients. The abdominal walls lose their elasticity and give to the palpating fingers a sense of doughy-resistance of irregular distribution. We can often feel an elongated tumor, painful on pressure, which runs above the navel, straight or transversely across the abdomen, and consists of thickened omentum; or we feel, in the neighborhood of the umbilicus, tumor-like masses of varying size, close to the abdominal wall. When the exudate has to a great extent been reabsorbed, we find in other parts of the abdomen tumor-like formations due to adhesions of the intestinal coils. The general appearance of the patients has changed for the worse; and their pallor and emaciation have become marked, the results of the continued fever that is usually associated with the development of cheesy changes in the tubercles.

Abdominal pain is rarely absent, but varies in intensity. Sensibility to pressure is usually present, but may be scarcely noticeable. Fever is present in all cases at some period, but varies according to the course of the disease. Constipation alternates with diarrhœa.

Thomayer's symptom may be of diagnostic importance; namely, the finding of dulness on percussion of the left side of the abdomen, with a tympanitic note on the right side. I have found these conditions present on several occasions.

I must also mention a physical sign associated chiefly with large exudations; namely, indolent and transitory reddening of the skin above the umbilicus. The sign may also be found when pus is present. Localized, encapsulated abscesses may perforate through the umbilicus (periumbilical phlegmon).

When improvement sets in, the fever disappears or lessens, and the exudation nodes and thickenings of the peritoneum become less and less. Sometimes rather quickly, but more commonly gradually and slowly, improvement sets in; and convalescence is followed by complete cure. Should the disease take an unfavorable turn, death may follow through gradual exhaustion; or as the result of intestinal

perforation (suppurative peritonitis), or from intestinal occlusion (formation of bands or shutting off of adherent loops of intestine).

When nodular tumors are present, differential diagnosis from sarcomatous and lymphosarcomatous new growths may offer some difficulties. The demonstration of tuberculosis in other organs may furnish a clue, or we may give tuberculin injections.

The paths by which infection reaches the peritoneum are by no means always evident, but vary much in individual cases. In the first place, we are inclined to consider the intestine as a likely port of entry. Considering the infrequency of diffuse tuberculous peritonitis, in spite of the existence of extensive tuberculous ulcers in the intestine, such an assumption is hardly justified in the majority of cases. Peritonitis may

FIG. 64.

Caseous peritonitis with moderate exudate. Great emaciation. Boy three years of age.

proceed from the lymph-nodes of the mesentery, the retroperitoneal, and the inguinal lymph-nodes (coxitis); but usually remains circumscribed.

The female genitalia assume considerable importance in the *etiology* of this disease, and their examination must not be overlooked. I recently saw, at the autopsy of a two and a half year old child that had suffered from caseous peritonitis and died of miliary tuberculosis, advanced tuberculous disease of the tubes; and found the corpus uteri transformed into a cheesy mass. Brüning has reported two similar cases, and Borchgrevink found four children under twelve years of age among ten patients affected with tuberculosis of the Fallopian tubes. In other cases, peritonitis is only a part of a general tuberculous "polyserositis." Peritonitis seldom occurs in connection with tuberculosis of the lungs. In quite a few cases we cannot discover the mode of infection. As yet, we are not in a position to decide whether we are justified in assuming for these cases hæmatogenous infection.

The **prognosis** of tuberculous peritonitis used to be considered unfavorable. This is by no means our present standpoint, when every year brings reports of recoveries and marked improvement in these cases, with or without operation. Naturally the prognosis depends on the extent of tuberculous involvement of the other organs. In childhood, peritonitis constitutes apparently the primary disease in a comparatively large number of cases.

Treatment.—The great question in the therapeutics of tuberculous peritonitis is, Shall we operate or limit ourselves to medical treatment? Whereas, a few years ago, the results of abdominal section were lauded to the skies; nowadays, the views, even of many surgeons, have changed. The Norwegian surgeon, Borchgrevink, has even made the statement that operative interference never helps, and only does harm. This change of view has come about from our observation of the frequency with which peritonitis (both the ascitic and the dry form) heals without operation. Our task must, then, be to place the patients in the most favorable conditions for recovery.

In the first place, rest in bed is of absolute importance. We frequently have opportunity to observe the favorable effect of rest in bed, when these patients are brought to the hospital. The ascitic exudate becomes absorbed with no other treatment; and the general condition improves, with eventual gain in weight. In the second place, proper nourishment is of great consequence. Since these children usually suffer from digestive disturbances and loss of appetite, the problem of their dietetic treatment may be extraordinarily difficult. Milk and foods prepared with milk; starch gruels, besides the infants' foods; plasmon; and similar preparations, must be tried. Some physicians ascribe especial value to carefully prepared dishes of meat; fresh beef-juice and the artificial foods prepared from meat. When persistent diarrhœa is present, the diet requires especial vigilance.

A prolonged sojourn by the sea contributes powerfully to success; but since the children from inland districts cannot often have this advantage given them, I would emphasize the fact that excellent results may also be obtained by a stay in mountainous districts under suitable conditions. Medium altitudes are to be preferred, as a rule; but even very elevated health-resorts, such as St. Moritz, Davos, Arosa, and Leysin (1500 to 1800 metres) are well tolerated by children that are not too much reduced in strength. The prejudice against sending children to these resorts is not justified. They may spend a large portion of their time resting in the open air with great advantage.

Inunctions of green or barrel soap, I use for *local treatment*. Like Heubner, I order the soap to be spread on thick, rubbed in, and allowed to remain for a quarter of an hour; then it is washed off. To avoid irritation of the skin, the treatment must be omitted every other day.

Applications of ichthyol also appear beneficial. Internally, I usually give thiocol, in doses of 0.25–0.5 Gm. (four to seven grains), three times a day.

According to my views, the value of *operative treatment* is now rather understimated; whereas, before, too much value was ascribed to it. Without doubt, many children improve rapidly after abdominal section, with disappearance of the ascites, fever and pain. When the ascites is very marked, the mechanical hindrance to respiration furnishes a definite indication. Most authors consider puncture of the abdomen to be of subordinate value, as compared with abdominal section. Since laparotomy is performed so much more frequently, there is not sufficient material to institute comparisons. Theoretically, it would appear that evacuation of the exudate by puncture ought to produce as good results as the same operation for serous pleurisy; but the conditions are essentially dissimilar. While the pleural cavity may be tapped without danger, in abdominal puncture for the relief of ascites the needle may penetrate adherent intestinal walls. On the other hand, the aseptic opening of the abdominal cavity is scarcely more dangerous than puncture and has an advantage in that we can study the extent of the lesions.

Operation must be advised in tuberculous peritonitis accompanied by exudation, when the latter does not permanently disappear on medical treatment. However, when there is no positive indication for surgical intervention, medical treatment should be given a fair trial. When no fluid exudate is present, the indications for operation are usually not so clear. We know that this dry type of the disease may heal without operation, so that some entirely reject laparotomy; whereas, others claim good results from it. When we are justified in assuming that tuberculous organs (tubes, ovaries) may be removed at the operation, a definite indication exists. Kocher has reported good results from such operations. Circumscribed collections of pus must be treated on general surgical principles; also complicating fistulæ, intestinal occlusions, etc.

Concerning the technique of the operation, most surgeons make a short incision in the linea alba, below the umbilicus; evacuate the exudate; and immediately close the abdominal cavity again. Care must be taken not to injure coils of intestine that may adhere to the abdominal wall. Professor Tavel (Berne) usually flushes the abdominal cavity thoroughly with a solution of vioform and normal salt solution.

TUMORS OF THE PERITONEUM

Benign and malignant tumors occur in childhood, and even in the newborn. Among the former must be mentioned cysts, which may be lymph-cysts, chylous cysts, or dermoid cysts. Other benign tumors are rarely encountered clinically. Malignant tumors are rare. Primary as well as secondary carcinomas, endotheliomas, and sarcomas may occur. The clinical symptoms resemble those in adult life.

DISEASES OF THE LIVER

BY

PROFESSOR M. STOOSS, OF BERNE

TRANSLATED BY

DR. CHARLES F. JUDSON, PHILADELPHIA, PA.

Introduction.—The size and weight of the liver in the newborn infant and in early childhood is great in proportion to the rest of the body. Birch-Hirschfield give the following figures:

Age.	Average body weight.	Weight of liver.	Per cent. of body weight.
At birth	2991 grams	127 grams	4.2 per cent.
6 months	3200 grams	197 grams	6.1 per cent.
1 year	5350 grams	312 grams	5.8 per cent.
2 years	8000 grams	346 grams	4.3 per cent.
3 years	9500 grams	453 grams	4.7 per cent.
4 years	11000 grams	535 grams	4.8 per cent.
5 years	12000 grams	480 grams	4.0 per cent.
7 years	18000 grams	638 grams	3.5 per cent.
10 years	25100 grams	830 grams	3.2 per cent.
In adult life	58600 grams	1624 grams	2.7 per cent.

The lower edge of the liver extends farther towards the umbilicus in the newborn and in early childhood than in later life. This is especially noticeable in the lateral region of the abdomen between the right mammillary and axillary lines. The reason for this low position is not the marked development of the liver at this age; it depends upon the configuration of the ribs, which take a more transverse course than in adult life and so leave more of the liver uncovered (Sahli).

These conditions must be considered in palpation and percussion. Since the lower edge of the liver can as a rule be readily felt, we ordinarily employ only palpation. Percussion becomes more important in later childhood. We percuss that part of the liver which lies next the abdominal wall, obtaining absolute or superficial dulness. Relative (deep) dulness gives us no certain results. The upper border of dulness (corresponding to the dividing line between the lungs and liver is found in the mammillary line at the upper edge of the sixth rib, just as in adult life. Percussion must be very gentle and proceed from above downwards.

JAUNDICE

Jaundice may occur in childhood under many different conditions. The jaundice of the newborn will be considered in another part of this work. Furthermore icterus may result from the action of various

poisons or drugs. It has been observed after treatment for tapeworm with the extract of male fern, also after the use of santonin. I have seen jaundice occur after the prolonged administration of lactophenin. Formerly icterus was observed after injection of tuberculin; with the small doses now used this is not likely to happen.

Acute infectious diseases may be accompanied by jaundice; this is not uncommon in pneumonia (bilious pneumonia), but this type is seldom encountered in childhood. In scarlet fever jaundice may be present in the mild type as well as in the septic cases, where it is an ominous symptom (Heubner). Icterus may also be a symptom of septic infection.

Gall-stones very seldom give rise to jaundice in childhood. Very few instances of cholelithiasis are on record in childhood. Those which have been reported ran their course with typical violent colic, in no sense differing from the adult type of the disease. Jaundice associated with acute and chronic inflammations of the liver and with tumors will be considered with these diseases.

CATARRHAL JAUNDICE

The most common form encountered is *catarrhal* jaundice, which results from extension of gastric catarrh, and is commonly due to overloading the stomach with improper food and drink. In many cases the inflammatory process extends from the duodenum to the biliary passages, which are both involved in the catarrhal process. Apparently such an extension does not occur readily, otherwise catarrhal jaundice would be much more frequent in young children who so often over indulge themselves.

It is well known that icterus is rarely associated with the gastro-intestinal catarrhs of infancy; even in enteritis which has led to ulceration of the duodenum I have never seen jaundice develop. In the case of older children jaundice rarely results from overeating in my experience. The ordinary catarrhal jaundice of childhood presents a similar picture to that of an acute infection.

INFECTIOUS ICTERUS

The occurrence of widespread infectious jaundice, epidemic in type has been repeatedly noted in early life. Sporadic cases are less frequent. This fact alone goes to prove the infectious nature of jaundice, occurring at this time of life. Epidemics have been reported from many different countries and I have at my command considerable data concerning those which broke out in Switzerland.

The city of Berne has been afflicted with various mild epidemics of jaundice. This was especially noticeable at the time of an epidemic of typhoid fever (from which disease Berne is usually free) which was

due to infection of the water supply (1898). I have also notes on small household epidemics where several children of one family were taken sick one after another with a febrile jaundice, associated with swelling of the liver and spleen. Beatenberg was visited with an epidemic of jaundice in 1898. Within three months fifty children who all went to the same school were taken sick. Three other schools escaped infection. A similar epidemic occurred in Cressier in Freiburg in the same year. Nearly all the children attending school were successively attacked. A general inquiry among Swiss physicians showed that there had been several small epidemics of infectious jaundice in that year and that in former years also the disease had been wide-spread, attacking principally the children. We must therefore admit then that jaundice in epidemic form is by no means infrequent in childhood.

The **source** of the infection cannot as a rule be determined; the physicians of Beatenberg could find no definite cause for the outbreak; the water supply was good, the weather warm and dry, and there had been no gross errors in diet. So many cases of infectious jaundice developed in Saxony (1888 to 1889) that they constituted a genuine epidemic, for which Meinert could find no cause. The simultaneous occurrence of so many cases in Switzerland in the year 1898 would seem to show that atmospheric conditions played some part in the causation of the disease. The effect of improper food in causing jaundice is illustrated by the following history of a family in Mettmenstetten (Switzerland) where six children were taken ill with jaundice almost at the same time.

The father who kept many bees had prescribed a honey cure for the children, which resulted after several weeks in digestive disturbances and jaundice. The children recovered promptly when the honey cure was discontinued. Two months later the honey cure was again begun; jaundice followed in three weeks time.

Most cases occur in children over three years of age. Younger children are also infected. In the Zurich home for rachitis at Ægeri within six weeks time four children from one to two years of age became jaundiced (1891). The febrile manifestations lasted from eight to ten days.

The **onset** and **course** of this disease are like one of the infections. The onset is usually sudden with fever, vague general pains and chills or vomiting. The stools may be loose or there may be constipation. Some epidemics may begin with angina, in others bronchitis is a constant feature. The yellowish discoloration of the skin and the mucous membranes usually appears on the second day, associated with other physical signs of jaundice. The liver is usually considerably enlarged, sensitive to pressure, as is also the spleen. In older children the pulse may be slow. This is not usually the case with

the younger ones. A trace of albumin is usually found in the urine. The yellowish color of the skin is often only indicated, but may be very intense a few hours after the onset of the disease. The fever generally disappears after a few days and the children make a prompt recovery. The jaundice also goes quickly in most cases. Occasionally we meet with protracted cases.

The disease usually runs a favorable course in our country (Switzerland); elsewhere the results have not been so favorable. Out of five hundred and eighteen cases thirteen died (Meinert), two of whom presented the picture of acute atrophy of the liver. Kissel had six fatal results out of 96 cases. Towards the end of the disease severe nervous symptoms (convulsions, loss of consciousness, coma) appeared, resembling cholæmia.

This type of the disease is closely related to *Weil's Disease* (icterus gravis) which must also be considered an infectious jaundice. In this disease, besides the high fever and marked nervous symptoms are often encountered hæmorrhages under the skin and mucous membranes. Adults are more frequently attacked by this disease.

Jaeger identified the bacillus proteus fluorescens as the *etiological factor* in a severe type of this disease. Whether proteus species directly invade the liver and the bile-passages, or whether they act through their toxines in the milder forms of Weil's disease, remains yet to be determined; so also is the part played by other micro-organisms (bacillus coli, etc.).

Attempts to agglutinate typhoid bacilli (Grünbaum, Zupnick, and others), the paratyphoid bacilli (Netter), with icteric serum have shown that icteric serum usually agglutinated more strongly than normal serum. Langer (Prague) obtained negative results in 14 cases, using Ficker's method of diagnosis.

Our **prognosis** must be made with care in infectious jaundice, though as a rule the disease terminates favorably in childhood.

Treatment is prinicipally dietetic. Fatty foods must be especially avoided and rice, barley or oatmeal gruel given freely, besides milk diluted with tea. In the latter stage of the disease wheat bread, white meat and preserves may be allowed. Constipation must be prevented and rhubarb is of service; but in some cases intestinal irrigation may be of decided help. Hydrochloric acid in small doses is valuable for its tonic effect. Should diarrhœa be marked and the stools become offensive, small doses of salol 0.25–0.5 Gm. (4–8 gr.) four times a day will be of service.

ACUTE YELLOW ATROPHY OF THE LIVER

The characteristic of this disease is the rapid diminution in size of the liver in a short space of time, from a few days to one or two weeks. These cases are occasionally encountered in childhood. The pathology, symptomatology, and the course of the disease are the same as in adults;

jaundice, high fever, marked nervous symptoms, convulsions followed by somnolence and coma as well as hæmorrhages are the predominating features. Objectively we find the region of the liver sensitive on pressure. Palpation and percussion demonstrate the shrinkage in volume of the liver. Simple jaundice may be present several weeks before the onset of severe symptoms.

A fatal termination is invariable in this disease. The etiology is not definitely known. Apparently the disease may be primary, or it may follow typhoid fever, erysipelas, osteomyelitis and general septic infections. Absorption of toxic products from the intestinal tract and syphilis have been considered etiological factors. In four cases Babes found streptoccoci and von Kahlden found bacterium coli in the capillaries of the liver; other investigators found no micro-organisms. Similar pathological changes are the result of various poisons, especially phosphorus.

DEGENERATIVE PROCESSES

1. *Cloudy swelling and fatty degeneration* only in rare instances give rise to clinical symptoms. There may be some demonstrable enlargement of the liver. Jaundice is nearly always absent. The etiological factors are essentially the same as in adult life, namely acute and chronic infectious diseases and disordered metabolism. Fatty degeneration is often found as the result of severe gastro-intestinal disease, in young children and especially in atrophic infants.

2. *Amyloid degeneration* of the liver is by no means frequent in childhood, in spite of the numerous chronic suppurative processes such as tuberculosis of the bone and glands, although many text books make the contrary statement. When the amyloid infiltration is marked, the liver may become very large, with the lesser grades there is no increase in size but the liver is heavier and firmer than normal. This disease presents no definite clinical picture.

ABSCESS OF THE LIVER

Abscess of the liver is a rare disease in childhood. Legrand could collect only 112 cases from the literature. Of these 31 were dysenteric abscesses (mostly from the tropics) 19 were traumatic, 15 due to appendicitis, 6 to typhoid fever, 10 were tuberculous, 13 due to worms, 2 followed phlebitis of the umbilical vein, 1 followed influenza, 9 were pyæmic and 6 were of doubtful origin. The effects of trauma, the migration of round worms and appendicitis are accordingly the most frequent causes of abscess of the liver in childhood. Those forms which have been brought about by ascarides deserve special mention.

Vierordt reports a very interesting case where the autopsy revealed the presence of ascarides in the bile duct, the hepatic duct and the

intrahepatic biliary passages with the formation of multiple abscesses. Ascarides were also found in the pancreatic duct; the tail of the pancreas was necrotic and had suppurated.

In general the **symptoms** are like those found in older persons; pain in the region of the liver, swelling of the liver, either upward or downward, protrusion of the whole hepatic region which may be very marked, and fever. In the absence of these symptoms the diagnosis may be extremely difficult.

The **prognosis** depends somewhat on the cause. It is more favorable for traumatic abscesses (12 recoveries out of 17 cases). Abscesses due to worms or following appendicitis are usually fatal.

Treatment consists of free incision of the suppurative foci.

CIRRHOSIS OF THE LIVER

Cirrhoses of the liver are not as frequently met with as in adult life. The etiological factors are essentially the same, but the chief of these, abuse of alcohol, is comparatively rare in childhood. It is easier for the cells of the liver to regenerate in childhood even after very severe injuries. Although severe alterations in the cells of the liver result from bad cases of scarlet fever, diphtheria and measles, we have no proof that permanent changes arise of the nature of cirrhosis. Evidently cirrhosis is the result of the combination of various factors. The most important varieties encountered in childhood are: (1) atrophic alcoholic cirrhosis; (2) hypertrophic cirrhosis; (3) cirrhosis due to circulatory disturbances; (4) cirrhosis from congenital obliteration of the bile duct. Syphilitic cirrhosis of the liver will be spoken of elsewhere.

1. ATROPHIC ALCOHOLIC CIRRHOSIS (LAENNEC'S CIRRHOSIS)

Most of the reported cases of this disease are from England where the poor children in the large cities are given brandy and gin at an early age, but the number of cases reported from other countries (France, Germany, Italy, Switzerland) is daily increasing. Hutinel believes that comparatively small amounts of alcohol may give rise to cirrhosis of the liver in childhood and that the period of alcoholic indulgences need not be long. The **duration** of the disease is shorter than in adult life, its **course** subacute. In the preliminary stage there is dyspepsia, loss of appetite, tympanites and constipation. Diarrhœa sets in at times and the children lose flesh. Then ascites appears with hypertrophy of the spleen and usually dilatation of the superficial veins of the abdomen (caput medusæ). The atrophy of the liver which develops insidiously may now be demonstrated if the ascites is slight or after abdominal puncture. Dulness on percussion is diminished, especially over the left lobe of the liver. Palpation may reveal the increased density of the liver and perhaps also irregularities on the surface. Jaundice is

absent or scarcely noticeable (subicterus). Later in the disease hæmorrhages may occur (hæmatemesis, epistaxis, melæna). Finally dropsical effusions appear and the child succumbs, often from œdema of the lungs.

All observers are agreed that the **diagnosis** is difficult. Our most difficult task is to differentiate cirrhosis from tuberculous peritonitis. In favor of the latter disease is its greater frequency. In those cases where the two diseases are associated (a not infrequent occurrence) the diagnosis offers unusual difficulties. Hutinel observed that cirrhosis of the liver in childhood was frequently associated with tuberculous

FIG. 65.

Cirrhosis of liver in a child six years old. Repeated evacuation of ten quarts of fluid.

disease of other organs, and this was also true of the cases recently reported by Beck, and Passini. The pathological findings are the same as in adult life.

2. HYPERTROPHIC CIRRHOSIS OF THE LIVER (HANOT'S CIRRHOSIS)

Hypertrophic cirrhosis is met with more frequently in childhood than the atrophic form. The etiology is still completely unknown. Hutinel, judging from the manifestations and course of the disease, classes it as a subacute infectious hepatitis, brought about by an unknown agent. Some cases may follow the acute degenerative processes. We can ascribe no importance to syphilis, alcoholism, malaria, or to the acute infectious diseases (scarlet fever, diphtheria, etc.) as factors in

the production of hypertrophic cirrhosis. Evidently accessory factors of whose nature we are ignorant are necessary. Some authors ascribed the disease to chronic gastro-enteritis, but we know nothing definite. In 2 cases of this disease described by Audénoud (one 14 months and one 16 months old) no marked digestive disturbances had gone before, nor had there been any acute infectious disease.

Chronic intensely marked jaundice with considerable enlargement of the liver and spleen characterizes the disease; ascites is not present or develops later. The cirrhosis runs a variable course; febrile attacks associated with pain about the liver alternate with pyrexia.

The liver is large, of a firm wooden consistency. Splenic enlargement may be so considerable that the organ can be felt to the right of the navel simulating the leukæmic spleen. We often encounter in children a very large spleen with relatively little increase in size of the liver, associated with joint deformities, especially of the fingers and toes. Arrested growth is a further characteristic of cirrhosis in childhood, says Hutinel. The children remain small and the limbs slender in striking contrast to the marked distention of the abdomen. Throughout the disease the liver remains large and jaundice persists. The fatal termination is reached after several years, but in infants the disease runs a more rapid course.

3. CARDIAC AND CARDIO-TUBERCULOUS CIRRHOSIS

Cirrhosis from failure of the circulation and chronic congestion presents peculiar features to which the French have called our attention: Hutinel has demonstrated the especial importance of this form of cirrhosis in childhood. The cause is chronic adherent pericarditis (with more or less complete obliteration of the pericardium), of rheumatic or tuberculous origin. Such cases have been encountered often in adults and described by F. Pick as "pseudocirrhosis of the liver due to pericarditis."

The liver has the typical "nutmeg appearance," with increased periportal connective tissue formation and cellular infiltration. In the cardio-tuberculous form the liver is always enlarged and perihepatitis is present. The pericardium is much thickened, resembling in some places a fibrous sack, with extensive adhesions, the latter often extracardiac. Pleurisy and tuberculous peritonitis as well as tuberculosis of the mediastinal lymph-nodes are regularly encountered.

Ascites and enlarged spleen are associated with the large fat, smooth or slightly granular liver, and the ascites is often the principal clinical feature. Other evidences of passive congestion are absent or of subordinate importance. The ascites is little or not at all affected by treatment with digitalis as in atrophic cirrhosis. It is probable that a common etiological factor gives rise to the affection of the pericardium

and the liver; whether the pericarditis is always primary is not certain. Chronic pericarditis, peritonitis and also pleurisy (polyserositis) may have a common origin, and the perihepatitis may lead secondarily to cirrhosis of the liver. Of 112 cases collected by Hess, 22 were under 16 years of age, and 16 under 19 years of age.

The disease which Curschmann designated as a sugar-cake liver belongs in this category. We have to do with a peculiar form of perihepatitis running an extremely chronic course. The peritoneal covering of the liver becomes changed in well-developed cases into a pure white sugar-cake-like mass, consisting of dense hard scar-like thickenings of the peritoneum. This white mass may be found on the peritoneal covering of the spleen, the anterior abdominal wall, and, at times may extend over the whole peritoneum. In 90 per cent. of all cases a more or less extensive connective tissue thickening and obliteration of the pericardium is present, as in cardiac cirrhosis. The liver itself shows no genuine cirrhotic changes; its surface appears rough and uneven. Ascites is always present and reaccumulates after abdominal puncture. The discovery of bands of thickened peritoneum will strengthen the diagnosis. The differential diagnosis of atrophic cirrhosis is often very difficult.

The **etiology** is unknown. Every age may be affected, several cases have been reported in children. The **prognosis** is absolutely unfavorable. The disease is progressive and terminates fatally.

4. CIRRHOSIS FROM CONGENITAL OBLITERATION OF THE BILE DUCTS

Congenital obliteration of the bile ducts is a not unusual malformation. The cirrhosis is brought about by simple stagnation of the bile, such as can be produced experimentally. In two of my cases the common bile duct was replaced by a fibrous cord. The gall bladder was absent. The liver was much enlarged and finely granular, the peritoneum much thickened with fibrous deposits, and the liver tissue showed cirrhotic changes. Some of the children are born jaundiced, sometimes the yellow tint appears first a few days after birth. The jaundice persists to the end. The abdomen becomes markedly distended. Severe toxic symptoms arise; convulsions, hæmorrhage from the navel and vomiting of blood, often as early as the first week. Umbilical hæmorrhage caused death after 14 days in one of my cases. Should the child survive longer, ascites appears; a breast-fed child whom I observed, survived for 6 months. In most cases this malformation depends on a primary defect of development, in other cases disease of the fœtus has been thought to be the cause.

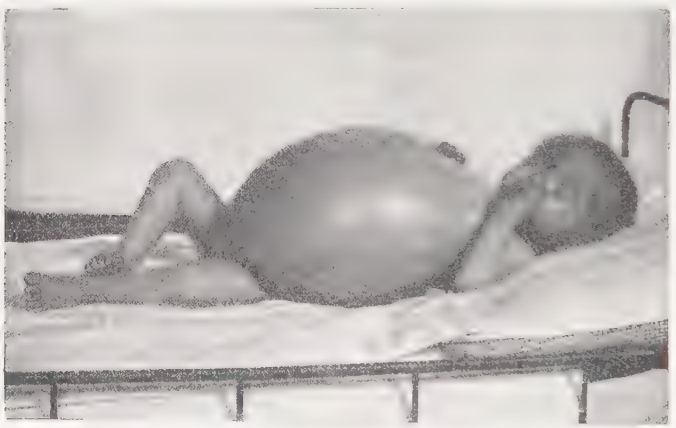
The **treatment** of cirrhosis must be directed against the causative factor when possible. If misuse of alcohol can be demonstrated we can do away with this factor and order unirritating diet, principally

milk. Should the cirrhosis depend on syphilis, or should this factor even be suspected we must institute antisyphilitic treatment with iodide of potash or with mercury. Extract of the liver has been given in France, according to Gaillard, with success in an advanced case of atrophic cirrhosis, d'Espine has seen marked improvement in the cardiac form of cirrhosis follow a sojourn by the sea.

Talma's operation, to relieve congestion by opening new paths for the portal circulation has been frequently recommended, and several times performed on children. I have the following case-report. (See Fig. 65, page 273.)

A four-year-old child was taken sick acutely in the fall of 1897 with fever, slight icterus and marked ascites. Repeated abdominal punctures were made the next year, and in the early months of 1899 ten quarts of fluid were frequently evacuated. Talma's opera-

Fig. 66.



Cystic liver.

tion was performed in June 1899. The ascites collected again after the operation, and twelve days later five quarts of fluid were evacuated by puncture. A second puncture five days later evacuated three quarts. In the subsequent period the ascites did not appear and the child felt well enough to visit school from January to May 1900. Then the ascites returned necessitating puncture in July (8 quarts) and August (14 quarts). After a second Talma operation, pneumonia developed and the child succumbed. The post-mortem findings were a very small, rough liver, only half the normal size, and a bundle of large veins at the point of anastomosis between the omentum and the abdominal walls.

The favorable result from the operation was unmistakable but not of long duration. Since the prognosis is bad, operation should be advised; there is always a chance of obtaining good results.

TUMORS OF THE LIVER

Benign and *malignant* tumors of the liver are encountered in childhood. Among the former we reckon angiomas and cystic degeneration of the liver. Fig. 66 shows a girl two years old with cystic liver, who became icteric shortly before death. The most prominent malignant tumors encountered are carcinoma and adeno-carcinoma, less often sarcoma. Steffen has collected 39 cases of primary malignant new growth of the liver in children. Several cases were in the newborn. I have observed adeno-carcinoma of the liver in a five-year-old boy with metastases in the lungs. Ascites was absent until shortly before death as in most of the cases reported. Jaundice also appeared only towards the end of life. Death followed hæmorrhage in the tumor. Parasites which may be found in the liver are distomum hepaticum, ascarides, echinococcus, cysticercus, and pentastomum denticulatum. The diseases to which they give rise are not different from those in adult life; these parasites are rarely encountered and are of little clinical importance with the exception of echinococcus and ascarides.

THE PATHOLOGY OF METABOLISM

BY

Dr. W. FREUND, of Breslau

TRANSLATED BY

Dr. CHARLES F. JUDSON, Philadelphia, Pa.

The consideration of those physiological processes which we call metabolism, whose aim is the measurement and valuation of the forces and energy developed from the food introduced into the system, for the manifold needs of the body, is of especial importance in childhood. At this epoch the necessity for the economy exists not only to supply new material to take the place of that consumed by the vital processes, but also to provide the means for the normal growth and normal development of the immature organism. Every disturbance of the normal course of metabolic processes in childhood hinders the normal functions of the body, and endangers also the normal development of the body, more especially in the first year of life when the general development of the body must take place quickly. When we consider on the other hand that at no other epoch of life is the organism liable to the same extent to disturbances of nutrition as in infancy, we must admit the vital importance of this subject. Unfortunately the confession must be made that our knowledge of the pathology of metabolism is scanty at the present time. The practical physician must know how limited and how few are the ascertained facts (based on correct theory) on which we can rely and how weak the links connecting these facts with the results of clinical observation. He will then be in a position to consider critically the various schemes and methods of feeding infants, which depend many of them solely on theoretical speculations and not on observations at the bedside of the sick child.

Since we are justified in assuming that the metabolism of the older child (under normal as well as pathological conditions) gradually approximates after a certain age to the conditions which hold good in adult life, whereas only infancy takes an entirely separate position in this regard, and since moreover the greater part of the investigations which have hitherto been made concern the first years of life, the following pages will consider in the main the conditions governing metabolism in infancy.

(*a*) PROCESSES OF METABOLISM WITHIN THE INTESTINAL CANAL (DIGESTION, ABSORPTION)

As we follow the passage of the food through the intestinal tract of the sick child, we must first of all consider what abnormalities occur in the digestive processes under pathological conditions. The aim of our investigations must primarily be to gain definite knowledge of the functional activities of the different secretions of the intestine, through the study of the digestive secretions which flow into it. The little that is positively known (in spite of very numerous investigations) is comprised for the most part in our studies of gastric digestion, which have been greatly facilitated by the introduction of the stomach tube. According to the original determinations of Wohlmann we know that the first appearance of free hydrochloric acid in the gastric contents of the healthy child at the breast is from one and a quarter to two hours after the taking of food, and reaches its maximum about two and one half hours after that time, whereas these times must be lengthened half an hour to an hour in the case of artifically nourished infants. We also know from universal experience that a diminished production of hydrochloric acid occurs in almost every constitutional disease in infancy.

This holds good not only for acute and chronic disturbances of nutrition of alimentary or infectious origin, but also for febrile diseases affecting other parts of the body, and manifests itself in this fashion, that a great number of sick infants show no hydrochloric acid or at least show no free hydrochloric acid, even in the last stage of gastric digestion. This knowledge is of special importance in practice, since it has laid the scientific foundation for one of our most important advances in dietetics; namely, the institution of definite intervals between meals for feeding healthy as well as sick infants. Associated with this condition we usually find delayed evacuation of the stomach and diminished motor-activity of the stomach wall. The fixing of definite intervals is designed to favor the appearance of free hydrochloric acid towards the close of gastric digestion and thereby make sure of the presence of an important antiseptic medium in the gastric intestinal canal. We can also demonstrate very easily in the test-tube the marked differences between cow's milk and breast-milk in their power to combine with hydrochloric acid, and thus explain the necessity to maintain longer intervals between feedings for the artificially fed child than are necessary when the child is at the breast.

On the other hand, hyperchlorhydria has been occasionally encountered. However we must admit that in some of these cases at least [in all of whom the mechanical factor of the pyloric closure comes into play], we have to do not with an actual over-production of hydrochloric acid but rather with an increased concentration of the same in the stagnant gastric contents. In any case the occurrence of hyperchlor-

hydria is of great importance in symptomatology and diagnosis (Knöpfelmacher, Freund, Ibrahim, and others). We know that the ferments of the gastric juice, the pepsin and rennet ferment as well as the ptyalin of the saliva, are practically always to be found even in the sick child under the most widely varying conditions. Thus we have at least no theoretical reasons for expecting results from the administration of these ferments and we must accept with great reserve favorable reports of their action (rennet, pepsin) since we are still so much in ignorance of their physiological importance in infancy. We know still less about the activities of the small and large glands of the intestine under diseased conditions than we do concerning the alterations of the gastric secretions.

The power of the liver to "neutralize" poisons has not yet been studied in the child. Keller found that urea was formed in normal quantity, even in very sick infants. The theory that the occurrence of light colored or whitish stools in infancy was due to acholia has been disproved by Langstein, who demonstrated the presence in the feces of urobilinogen (a colorless product of biliary coloring matter in a more advanced stage of reduction).

We have some reason to believe that there may develop functional failure in digestive power of the pancreatic secretions, since the investigations of Gillet and von Jakubowitsch have shown that the diastatic, peptonizing and fat-splitting functions may be lost in certain diseases. [According to Zweifel, Korowin and others the saccharifying ferment is not present in the pancreatic juice in the first months of life.]

This is the extent at the present time of our theoretical knowledge of the forces which control the digestion and absorption of food in pathological conditions of early childhood. Putting these facts together we hardly seem justified in assuming that we will encounter decided disturbances of digestion and absorption of the different food stuffs in the gastro-intestinal canal. Nor do the results of our direct investigations of the processes of digestion favor the assumption, which for a long time predominated in the pathology of childhood, namely—that disturbances of nutrition at this time of life were substantially identical with disturbances of absorption of important food constituents.

We know from a series of more than forty experiments in metabolism (Bendix, Lange and Berend, Freund, Keller, Steinitz and others) that there is a *fairly good absorption of the nitrogenous substances* in the diet, even in the case of very sick children. It is true that the cases investigated were mainly subacute and chronic in character, whereas somewhat larger amounts of nitrogen are excreted in the feces during acute attacks of diarrhœa. In the latter cases it is possible that a considerable portion comes from the nitrogen-containing intestinal secretions and not from the food.

In pathological conditions the *absorption of carbohydrates* plays a more important rôle than the absorption of nitrogen. We know that children in the first months of life do not assimilate starchy food as well as older children (Heubner, Carstens) and the more complicated the food mixtures the more difficulty exists with the digestion of starches (Hedenius). We know moreover that in severe disturbances of health the splitting up of milk-sugar may not occur in the intestine (the younger the infant the more likely this is to happen); the latter is then absorbed unaltered and reappears in part in the urine on account of its relatively low limit of assimilation (Gross, Langstein and Steinitz).

Concerning the *disposal of fat* in the intestinal tract of sick children we know unfortunately not as much as we should like, considering the importance of the subject from the clinical standpoint. The question comes up here, whether considerable quantities of the fat taken in with the food may not under certain circumstances pass through the intestines unutilized; also we must consider the form of combination in which the fatty acid radical occurs in the villi; whether in the form of neutral fat, of free acid, of soluble or insoluble soaps, and what proportion these components bear to one another. In the first case we are confronted with the partial loss of an important food constituent. The few estimations of fat absorption which have been made in sick infants allow us to conclude that at times a considerable portion of the fat escapes absorption, but metabolic investigations have not yet determined losses of fat of sufficiently marked degree to jeopardize nutrition. From clinical observations however we know that cases are not uncommonly encountered where there is a great excess of fat in the feces. The inspection of the stools is of no practical value, nor is the estimation of the percentage of fat in the feces (which Biedert advised in cases of diarrhœa) sufficient to determine this question, since in these cases the amount and the nature of the other constituents of the stools is of more importance than the absolute amount of fat or the per cent. to which it is absorbed.

Another important question is the mode of combination of the fat in the feces, since this has an influence on the metabolism of the salts in the intestinal tract. For it is only in that rather unusual condition where the amount of neutral fats is relatively increased (Biedert's "fat diarrhœa" in the true sense of the word), that the excess of fat eliminated is without influence (ante portas) on the metabolism of the salts; whereas in that very common condition of the stools resulting from increased formation of soaps, which can be usually recognized by the naked eye, the alkalies and earths needed for this purpose are withdrawn and do not reach their usual destination. We will discuss the consequences of this process in another place. A third possibility, the predominance of free fatty acids in the fecal contents, plays a part in the production of the so-called acid dyspepsia of breast-fed infants, described by Raczynski.

We possess few data concerning the *excretion of mineral salts* in the feces of sick children, and these bear little or no relation to any definite pathological condition. An exception may be made for the excretion of alkalies, and of lime. Schkarin found the latter in increased amount in the stools in febrile conditions; his results are of much interest but require further confirmation.

Our knowledge of the conditions regulating the digestion and absorption of food-products within the intestinal canal would not be complete without some reference to the processes of *decomposition resulting from the presence of bacteria* in the intestinal tract. In the healthy breast-fed child fermentative processes predominate over those of putrefaction. In the healthy child who is artificially nourished, putrefactive processes to a limited extent are always demonstrable in the intestine; under diseased conditions the evacuations usually manifest from their offensive odor the considerable degree of putrefaction present (even when the infants are breast-fed). We must assume that the intestinal secretions furnish the material which undergoes these changes, for we know that milk is not liable to putrefaction, in fact milk-sugar may to a certain extent hinder putrefaction. Leaving now the field of strictly proven facts we know that the amount of the intestinal secretions is increased by artificial feeding and by certain diseased conditions, both of which promote putrefaction, all the more since the antiseptic action of the gastric juice is checked and limited under these conditions. In the healthy breast-fed child, fermentation exceeds putrefaction at all times, since the intestinal secretions are less active and the antiseptic action of free hydrochloric acid reaches its full development. A certain symptomatic importance must then be ascribed to the secondary process of intestinal putrefaction in the infant's intestinal tract; whether the products of putrefaction can directly influence or injure the organism, is not yet definitely known.

The finding of products of intestinal putrefaction in the urine of the infants helps to substantiate what has just been said. Indican is always absent from the urine of healthy breast-fed children, is not often found in the urine of artifically fed infants, but is frequently present in acute and chronic disturbances of nutrition. What we know about the ethereal sulphates in the urine during the first year, corroborates this view. The relation of the biliary coloring matter to intestinal putrefaction may aid in diagnosis. Bilirubin is found in the feces where there is no putrefaction (healthy breast-fed child); when there is putrefaction its reduction to hydro-bilirubin (urobilin, stercobilin) is favored (Schmidt's sublimate reaction—Schikora). In older children on a mixed diet as in adult life, we find under normal conditions intestinal putrefaction, hydrobilirubin in the stools, and decomposition products in the urine.

(*b*) METABOLIC PROCESSES BEYOND THE INTESTINAL WALL (ASSIMILATION, DISINTEGRATION)

In the metabolism of infancy there is one fact of great importance that characterizes it, namely, that the processes of growth normally bring about assimilation and retention of the materials in the food necessary for the development of the body. We must, therefore, investigate to what degree pathological conditions can influence the normal *retention of infant food* constituents. Many such investigations have been directed to determine the fate of the nitrogen in the child's food, and give the apparently paradoxical result that under almost all conditions, even in sick infants whose body weight is at a standstill or who are losing weight, a retention of nitrogen nevertheless results. This is in agreement with Camerer's observations that even atrophic infants show a growth in length and indicates the extraordinary intensity of the stimulus to growth within the body. The misproportion between this retention of albumin and a standstill or loss of body weight indicates plainly that other food-stuffs must pass unutilized through the body, and we are now in a position to state definitely which food constituent is primarily at fault. The trite statement that a child which is losing in weight or which is only holding its own becomes thin (that is, becomes poor in fat), has been confirmed by Steinitz's analyses of the total ash from the bodies of infants. The only marked difference in the chemical composition between the body of the healthy child and that of the infant who had died from severe illness, is the notable difference in the fat content.

What we know from metabolism experiments on the *retention of water* in the body agrees well with our clinical experience. Whereas the physiological assimilation of water amounts to 60 per cent. of the total assimilation, according to Camerer, and whereas this proportion is the same in infants who are gaining regularly in weight, under pathological conditions we find frequently very great changes in the body weight, either up or down, which can only be brought about by corresponding alterations in the watery content of the body,—for this reason, that the assimilation and excretion of the other constituents could not take place to the same extent in the same time. Our a priori assumption is herewith confirmed by exact investigation (Freund).

Whereas the phosphorus in milk (Keller) is usually retained well even by sick children (especially that of women's milk), and its assimilation runs more or less closely parallel to that of nitrogen, the chlorides on the other hand are apt to vary with the alterations in the water content of the body (Freund). Alkalies can be excreted by the intestine in such large amounts as the result of a diet containing plentiful amounts of fat, that the body may lose a considerable part of its alkali.

In febrile conditions, as we have already mentioned, the body may

lose lime in excess. We have little knowledge of the causation of other anomalies in the retention of lime salts, the end results of which are seen in the defective ossification of the rachitic skeleton as well as in the diminished content in lime of the brain in the condition known as tetany (according to Quest's investigations, which require confirmation).

The *processes of disassimilation* in the body, the so-called intermediary metabolism, also interest us from the pathological standpoint. Of these we assume rather than know that they deviate from the normal in the sick child. The sum of the processes of oxidation which bring about disassimilation, has been studied in chronic diseases of infancy from two standpoints. First of all the possibility exists that excessive heightening of oxidative processes interferes with the normal processes of assimilation and so creates conditions akin to those in infantile atrophy. The hypothesis which Bendix put forth with regard to these cases, has not been confirmed by the investigations hitherto made of the excretion of carbonic acid by atrophic infants. On the contrary Rubner and Heubner found no decided deviation from the normal, and Poppi even found a diminished excretion of carbonic acid. In the second place we have good reason to believe that there is a diminution of the normal processes of oxidation in severe cachexias in infancy, and certain facts seems to substantiate this view. Pfaundler has shown that there is functional loss of the oxidizing ferment of the liver in certain cases; moreover we know that the power of oxidizing benzol into phenol is very much diminished in the very sick infant (Freund). Recent experimentation in the same line by Ludwig F. Meyer has demonstrated diminished capacity of the infant organism to oxidize further the phenol taken into the body. In this place we must mention that French authors have ascribed an important rôle to the process of diminished oxidation (bradytrophy). These cases are characterized by an excess of uric acid in the circulation and a great variety of pathological phenomena, the so-called *"Arthritism"*. On the basis of a critical study by Göppert we must consider that the pathological-chemical basis of this constitutional disease remains still in the realms of speculation.

Langstein and Meyer have studied qualitative disturbances in the course of the processes of oxidation in older children, investigating the excretion of acetone bodies in febrile diseases and in conditions of inanition, especially where there was carbohydrate insufficiency. From these experiments one fact appears characteristic for childhood that the disturbances of the intermediary metabolism which lead to the *excretion of acetone bodies*, proceed from the same causes as in adult life; but occur very much more readily in childhood. A further peculiarity of infancy, according to these authors, is that the pathological increase in excretion of acetone takes place mainly through the expired air, and not as in adult life through the urine. Hüssy found in his experiments,

which are not yet published, that the excretion of acetone did not invariably follow this rule. The condition of *cyclic vomiting* with fever without organic disease (acetone vomiting) has been much discussed of late in French literature. In this condition acetone is perhaps the specific agent, it at least appears in large amount in the urine; on the other hand the diagnostic importance once ascribed to Legal's test for the differentiation of etiologically different throat inflammations has not been confirmed. The meaning of acetone bodies in the metabolism of infancy will be mentioned in the following pages.

Let us now leave the subject of the essential intermediary metabolism and consider the fate of the *mineral salts*, which are combined with the food stuffs and other constituents of the body, are involved in their disassimilation, and are excreted through the urine. One fact stands forth preëminently in this line of work, which has not only guided the investigations of pathological metabolism, but has especial importance at the present time when we consider the metabolism of salts. It also has a practical value for the study and successful management of the diet of sick infants. Keller found in the year 1894 that the urine of sick infants contained remarkably large amounts of *ammonia*, so much that in extreme cases the ammonia-nitrogen at times equalled nearly 50 per cent. of the total nitrogen excretion, or (more tersely expressed) that the so-called ammonia coefficient could rise almost to 50.

The most probable explanation for this remarkable fact was given by Thiemich, who succeeded in demonstrating an advanced degree of degeneration of the liver in infants, and in some children who had excreted during life these very large amounts of ammonia, whereby the possibility was suggested that the cause of the increased excretion of ammonia lay in the diminished power of the liver to form urea. Direct investigations, however, proved this supposition to be incorrect and showed that even very sick infants were able to transform the ammonia salts introduced, into urea. A large series of observations, and the well known property of ammonia—to appear in the urine in the company of acids,—led to this conclusion: increase in excretion of ammonia could be brought about by increase in excretion of acid products of metabolism. Hijmanns von der Bergh proved by the use of the so-called Schröder-Munzer criterium that this view was correct, since by the administration of alkali the previously high excretion of ammonia could be reduced to nothing. But whence came the quantities of acid, for whose saturation the organism is forced to manufacture such enormous quantities of ammonia, since the existing supply of alkali at the disposal of the body would by no means suffice for this purpose? A large number of experiments were carried out at the Breslau Clinic to decide this question, as to the influence exerted by the diet upon the excretion of ammonia in sick infants. They led to this definite result,

that the administration of fat brings about high excretion of ammonia, whereas the removal of fat from the diet causes its disappearance. The question still remained unsolved as to the nature and origin of the acids in question. The view was held for a long time by the pupils of the Breslau School, that analogous to diabetes there was an increased or abnormal formation of organic acids in the intestines, or in the intermediary metabolism, and that the cause of their incomplete combustion must be ascribed in part at least to a diminution of the normal oxidizing powers of the organism.

Steinitz's studies of the metabolism of alkalies showed that a genuine *acidosis* of this type does not ordinarily occur, and that the reason why increased fat in the diet led to greater excretion of ammonia by the kidneys, was altogether different.

Steinitz showed that the result of the introduction of definite amounts of fat into the intestines was increased formation of alkaline soaps; the alkalies required are thus prevented from reaching their normal destination (in the body); ammonia must be supplied to neutralize the usual inorganic acid end-products of metabolism, and therefore appears in the urine in increased quantity, whereas the alkalies are excreted in the feces and sometimes in such quantities that the balance of alkali in the body may remain constantly negative.

A second cause for the increased ammonia is the greater absorption of phosphoric acid from a diet rich in fat. Even when considerable quantities of lime-soaps are formed in the intestines, an increased excretion of lime through the intestines does not follow, but the formation of calcium phosphate (which is absorbed with difficulty) does not apparently take place to the same degree. A greater amount of phosphoric acid is absorbed under these conditions, possibly already combined with ammonia, in which form it is excreted in the urine (Freund).

The question is not yet decided whether, in addition to the derangement of mineral salt metabolism due to the withdrawal of alkalies, we may not also encounter sometimes a genuine acidosis, brought about by the increase of organic acids in the circulation; the previously mentioned investigations of acidosis in older children make this seem probable.

Even if the condition known as "*acid intoxication*" has not taken on more definite shape and form as the result of further studies, it led Czerny and his pupils to a thorough investigation of the question of ammonia excretion and has furnished valuable results from the clinical standpoint. Clinicians soon learned to avoid giving too large amounts of fat, and recognized the great advantages inherent in a diet rich in carbohydrates and low in fats in certain disturbances of nutrition in infancy. One knowledge of the harm which may result for the healthy infant in consequence of a diet which is persistently too rich in fats, is amplified by the recognition of the changes which follow in the metab-

olism of the mineral salts. Prophylaxis and treatment thus obtain a firm footing. Here we have at least one example of the harmful results of one form of overfeeding, where light has been thrown on our empiricism as the result of exact investigation. True, we know from the clinical standpoint the influence of too much proteids or starch in the diet; but from theoretical considerations their explanation is difficult.

A few words must be devoted to the subject of the amount of energy needed for its development by the infant. Rubner and Heubner made the first complete investigations of infant metabolism (including those substances which are excreted in gaseous form) which were directed to the quantitative determination of the energies remaining in the body as the result of the utilization of the food stuff; in other words they tried to establish a balance of energy. The details of the experiments cannot be discussed here. Heubner concluded from his observations of the food requirements of infants that the work of digestion was much more considerable when the child was artifically fed than when nursed at the breast.

On this basis he suggests an explanation for the occurrence of infantile atrophy. The work of digestion may be abnormally increased (according to Heubner) by congenital weakness of the intestinal tract or that produced by disease, so that of the calories introduced so large an amount is required for the work of digestion,—hence is lost for the body since the heat is dissipated,—that not enough remains for the work of assimilation; in fact some of the body substance needs to be disassimilated to furnish the energy required. In view of the clinical facts these theories are very plausible and interesting, they still lack absolute scientific proof of their correctness.

We must now consider the practical importance in the estimation of the food-requirements of the sick infant of Heubner's *quotient of energy*. First we must remember that we should not judge a food (for infants or adults) solely by its dynamic value, but that the form in which we give the required energy, to the sick infant is all important. A child injured by overfeeding with fat will not recover on a mixture containing much fat, even if it represents a sufficient number of calories; the first indication here is to change to a diet with low fat and more carbohydrate, afterwards the quantity of energy required may be regulated.

Lately Czerny and Keller have suggested the classification of the disturbances of nutrition in infancy on a new basis, and have thereby rendered practical service to the clinician. These authors sought to establish clinical pictures of disease based on pathological-chemical processes (milk-food injury, starchy-food injury, albuminous-food injury, etc.); and even if the latter must be considered still only as probabilities, they constitute a framework for our future investigations into the pathological-chemical causes of disorders of nutrition in infancy.

INTESTINAL BACTERIA

BY

DR. E. MORO, OF GRATZ

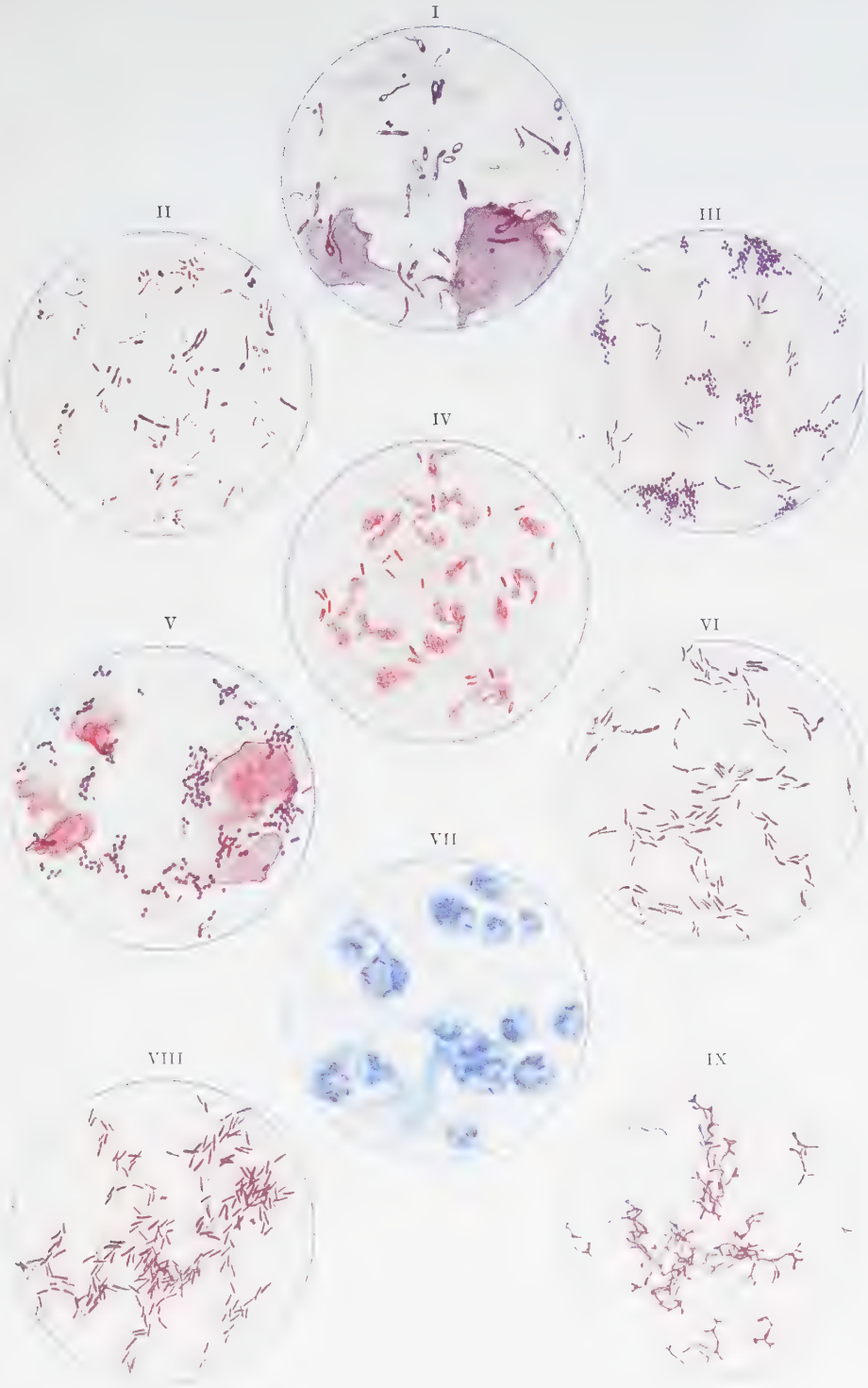
TRANSLATED BY

DR. CHARLES F. JUDSON, PHILADELPHIA, PA.

IN the nature of things, the bacterial contents of the intestines are most easily investigated when the food is of uniform character. The lively interest in the investigation of the etiology and pathology of infectious inflammations of the intestine in infancy has led to a thorough going study of the intestinal flora in the infant. After Robert Koch had delighted the scientific world by the discovery of plate culture and by the introduction of solid nutritive media, and so improved bacteriologic technique, Escherich wrote the first great work on the intestinal bacteria of infancy and gave a scientific basis to the study. Further contributions were rapidly made by Escherich and his pupils, who concerned themselves chiefly with the relations between certain kinds of bacteria and the origin of acute digestive disturbances in infancy, and with the biological properties of bacterium coli commune. Out of the large number of more recent investigations, only those of Tissier, in the year 1900, deserve to be especially mentioned; since these enriched our knowledge, and for the first time plainly proved the great importance of anaërobic methods of culture in the study of intestinal flora. Subsequently, the use of this method furnished valuable discoveries; and it is now indispensable for a correct judgment of the physiological conditions.

The end aimed at is to discover what forms of intestinal bacteria have to do with the causation of certain intestinal disturbances in infancy. It is clear that on this basis the most valuable contributions may be made to the rational therapy and prophylaxis of intestinal disease in early life. By this I mean not only those processes which Escherich has collected under the name of ectogenous infection, but much more the endogeneous fermentations that occur in the intestinal canal, which Escherich has called chyme-infection; and which, under the picture of an intoxication, play a predominant rôle in the pathogenesis of acute and chronic disturbances. The recognition of the latter—that is, the endogenous infections—is much more difficult than is the recognition of the former, or ectogenous infections; for many facts are now at hand to demonstrate the frequency of ectogenous infections.

PLATE 46.



BACTERIOLOGICAL PICTURE OF THE INTESTINAL BACTERIA.

I. Smear from meconium.
II. Smear from a normal cow-milk stool.
III. Smear from a breast baby suffering from acute enteritis.
IV. Smear from mucopurulent stool in colitis.
V. Smear from stool of acute enteritis in artificially-fed infant.
VI. Smear from a normal breast-fed infant.
VII. Smear of pus from a case of dysentery.
VIII. Smear from an artificially-fed baby suffering from acute enteritis.
IX. Smear from anaerobic culture of the bacillus bifidus grown on glycerin sugar agar.

Successful investigations of these processes presupposes, naturally, in both cases, a knowledge of the normal physiological conditions. It is only by following this method that we can reach our goal. The chief methods in studying intestinal bacteriology are microscopic investigations of the feces in stained preparation and culture. The Weigert-Escherich stain is the best.*

The intestinal flora may be studied to great advantage in the stained preparation, which allows us to recognize easily a condition varying from normal. The determination of the predominating types is possible after some experience with the microscope, so far as we have to do with the characteristic morphological types. When we have to make only a superficial examination, the preparations give valuable results which exceed in clearness and in extent those given by culture-methods.

When the result is not clear, and when we wish to study certain definite types further, microscopic investigation must be amplified by culture. It should be emphasized that in doubtful cases culture methods are of value only when different methods of growth are used, of which the most valuable are the anaërobic method, culture on acid nutritive media, and culture on media that have been enriched (for example, on milk and egg albumin under aërobic and anaërobic conditions); cultures may be made from pasteurized feces, or we may use combinations of the foregoing methods. Otherwise, one is liable to fall into the mistake of becoming elective and one-sided; and this may lead to dangerous conclusions.

A further problem is in testing the biochemical activities of isolated bacteria with reference to the food material present in the intestine. If we suspect the etiological importanee of a microörganism, animal experimentation may be used to decide the question. Then, in addition to the usual methods of infection, the test of intoxication with the corresponding bacterial filtrates may be made. Animal experimentation has here only a limited value, since harmless intestinal saprophytes may have a pathogenic action; whereas, animal experiment with germs that notably call forth acute digestive disturbances may fail one completely. On the other hand, other factors, which will be mentioned later, speak much more strongly in the individual cases for the pathogenic importance of certain kinds of bacteria.

* Reagents Required:

1. *Aniline gentianviolet*, an especially-prepared mixture of (a) a 2.5 per cent. watery solution of gentian-violet and (b) alcohol-aniline oil, 11: 3, in the proportion of 8.5 to 1.5.
2. *Watery iodine-iodide of potash solution*, 1: 2: 60.
3. *Aniline oil* and *xylol*, equal parts.
4. *Xylol.*
5. *Watery solution of fuchsin or safranin.*

Technique: The preparation, which is fixed on the object-glass, is, first of all, covered with solution No. 1. After ten seconds, this color is allowed to run off; and the cover-glass is dried with filter-paper. Then No. 2 is dropped on, and the glass is again dried with filter-paper. Then the preparation is decolorized with No. 3, the solution being poured on freely until the disappearance of the excess of coloring matter occurs. Directly afterward, No. 4 is poured on. After drying over the flame, make a contrast stain with No. 5 (for bacteria that do not stain with Gram's method). Wash off with water, dry, and examine in oil.

THE INTESTINAL FLORA OF THE INFANT UNDER PHYSIOLOGICAL CONDITIONS

The intestinal contents in the newborn child are free from germs; but infection of the intestine follows very quickly (within four to ten hours after birth), so that we can demonstrate microscopically bacteria in the second or third evacuation of meconium. The preparations of meconium show a very characteristic picture, peculiar to this time of life (Plate 46, Fig. I). The meconium flora are distinguished by manifoldness of form, but comparatively small number; as well as by the constant occurrence of bacteria containing spores. Of the latter, we especially note rods that take the stain poorly, carrying at one end an oval, shining spore. These have been called by Escherich "little-head" bacteria; and their narrow form has been compared to that of spermatozoa. Besides these, we find, usually, plentiful cylindrical rods, containing large spores. In some preparations, the spore-bearing bacteria predominate; in others, long, rather thick, partly stained bacteria, at times some of them wave-like and slightly bifid at the ends, are in the foreground. Their appearance precedes that of the spore-bearing bacteria. We must emphasize, besides this, the frequent presence of different kinds of cocci; and the regular presence of oval, short rods, not taking Gram's stain.

When the child is put to the breast and the first milk-stool appears, the vegetation alters suddenly. The previous flora make place for new varieties. We find thin rods predominating, usually pointed at both ends, which stain with Gram's method. These have a tendency to group themselves as diplobacilli, and sometimes show definite branchings. These bacteria multiply very rapidly and completely predominate in the field of the microscope (Plate 46, Fig. VI). I have proposed the name of the *physiological fecal flora* of infancy for this distinct type of flora in the stools of the breast-fed infant.

So long as the child is at the breast, and is healthy, the character of the picture does not change. With the introduction of artificial nourishment, a typical change of flora appears, however. The one type of vegetation soon entirely disappears, and in its place we have the picture of fecal flora of the child fed on cow's milk (Plate 46, Fig. II), which, in contrast to the previous picture, is characterized by a great multiplicity of species and their polymorphous character, as well as by the predominance of bacteria negative to Gram's stain. The following laws may be deduced from these microscopic findings:

1. *The intestinal flora depend upon the kind of food taken by the infant.* If the newborn child is fed from the beginning with cow's milk or in any other artificial way, it will never have the physiological flora in its intestine; but if an artificially-nourished child has human milk

given it at a later period, the physiological flora will appear in the feces after two or three days, with absolute regularity. The knowledge of this interesting fact gives the investigator the opportunity to decide, from a study of the feces alone, whether the child is nourished naturally or artificially.

2. *The microscopic picture of physiological intestinal flora is uniform and constant.*—The occasionally encountered cocci and bacteria negative to Gram's method in the stool of the nursing infant are present in such a small number that they can never, under normal conditions, influence the uniform picture of the flora. The results of culture are, to a certain extent, in conflict with the results obtained by microscopic investigation; for instance, the more sensitive method by culture shows us a larger number of types than we can expect to find by the microscopic method, and shows that the greater number of bacilli in the stool of the nursing infant are identical with the bacillus bifidus communis (Tissier). The bifidus is a strictly anaërobic bacterium, and is most easily cultivated on glycerin-sugar-agar. The most marked characteristic of its many morphologically interesting variations in growth on culture-media is the formation of ramifications resembling the antlers of a deer (Plate 46, Fig. IX). Besides this chief representative of the physiological fecal flora, Tissier obtained regularly from the stools of nursing infants three other types; the bacillus coli communis, the bacillus lactis aërogenes, and the streptococcus Hirsh-Libmann. To these I have lately been able to add three obligatory intestinal bacteria of nursing infants: the bacillus acidophilus, the "little-head" bacterium, and the motionless butyric-acid bacillus—the latter confirming Passini's findings. The motile butyric-acid bacillus and the putrefactive bacillus putrificus coli Bienstock (both markedly anaërobic) are also frequently, but not constantly, present in the stools of nursing infants. Altogether, I have been able to isolate nineteen different varieties from the stools of healthy nursing infants. In spite of this relatively large number, I am conscious of the fact that I have by no means exhausted the number of bacteria inhabiting the intestines of the breast-fed infant.

This is even more true of the intestines of the *artificially fed infant.* The microscopic picture, which is characterized by great richness in variety and form, indicates this definitely. The study of intestinal bacteria does not require so much the complete and systematic discovery of the bacteria present as the selecting from among the large number of saprophytic and transitory inhabitants of the infant's intestine those forms that are of importance for physiological processes in the intestine. The predominance of the colon group and the presence of bacteria having proteolytic action is especially marked in the feces of the artificially fed infants. The bacteria in the meconium, originally

described, were previously considered accidental inhabitants of the intestines, only demonstrable in the stools during the meconium period before they had been driven out by the germs taken in with the food; but my latest investigations have shown that a large number of the so-called meconium bacteria are identical with the organisms subsequently found in milk stools. The difference between them is simply external, affecting the form of growth; since the meconium furnishes an unfavorable medium for the growth of the entering germs, and compels the formation of spores and permanent types. Thus we find already present in meconium the bacillus bifidus, the bacillus coli communis, the butyric-acid bacillus, the above described "little-head" bacterium, and the bacillus putrificus—all forms of bacteria that are later found in the milk stool and continue to vegetate in the intestines after that period. The meconium prevents the indiscriminate migration of ubiquitous germs into the intestine of the newborn, and allows only those bacteria to enter that have a specific biological affinity to the intestines and their contents. Thus the specific infection of the intestines with the obligatory bacteria of the intestine is accomplished even in the first days of life. With the appearance of the first stools after nursing, the bifidus multiplies rapidly. When artificial nourishment is instituted, on the other hand, the colon bacteria predominate; and all other forms become of little importance.

3. The occurrence of a *changed vegetation in the feces* is, under normal conditions, not so much the result of new bacteria introduced with the food as the expression of an elective, one-sided multiplication of one or the other type of bacteria already present in the intestine. This, also, would explain the alteration of the intestinal flora as the result of a decided change in artificial feeding. A diet rich in starch favors the development of the saccharolytes; food rich in albumin, the vegetation of the proteolytes. The possibility therefore exists of restraining intestinal putrefaction by giving starch plentifully (Escherich).

I have demonstrated, by investigating bacteriologically immediately after death various portions of the intestine of breast-fed children showing no intestinal disease, that the different forms of bacteria are not distributed haphazard in the intestines but that their distribution follows certain laws. Especially remarkable is the fact that the upper parts of the intestine, particularly the small intestine, are nearly free from germs. The amount of bacteria does not become considerable until the cecum is reached; and from there, it increases steadily. Whereas the bacteria of the colon group predominate in the duodenum and the lower part of the ileum, the bifidus vegetation suddenly appears in the cecum, and in the colon already predominates over all other types of bacteria. The flora of the cecum, on the contrary, offers a more or less polymorphous picture. This is due to the frequent occur-

rence of the butyric-acid bacilli and other spore-carrying anaërobes in this section of the intestine. From these results it follows that the investigation of the feces in the stool gives no satisfactory information concerning the higher portions of the intestine, and that we must distinguish between the ideas: fecal bacteria, and intestinal bacteria. The quantity of bacteria excreted with the feces is enormous. From 20 to 30 per cent. of the total nitrogen in the stool of the breast-fed infant is due to bacteria. The majority of the microbes excreted are probably dead, but our information on this subject is generally defective, because the investigators did not always consider the anaërobic and acidophile forms.

The biochemical activity of normal intestinal bacteria in the infant expresses itself chiefly in decomposition of the food constituents and the food residue, of the nature of fermentation and putrefaction. The nitrogen withdrawn from the food remnants and built up into bacteria is, in all probability, more than off-set by the absorption of dead microbes in soluble form by the intestine.

The assistance that intestinal bacteria furnish to the process of digestion can be only small, so far as concerns the taking up of the food stuffs and their transformation into directly absorbable substances. Nevertheless, the assumption that intestinal bacteria, in a hitherto unexplained fashion, directly or indirectly take part in the process of digestion and favorably influence nutrition, cannot be altogether rejected. Experiments have demonstrated that newborn animals brought up with sterile surroundings and on sterile food remain decidedly backward in development as compared with control-animals, and show the influence that the intestinal flora exert upon nutrition. In fact, some animals could not be kept alive without intestinal bacteria (Schottelius, O. Metschnikoff, Moro, Nuttall, and Thierfelder).

Fermentation and putrefaction are antagonistic processes; that is, we cannot have putrefaction in a medium that is undergoing fermentation. Both processes are caused by the presence of specific bacteria. The energetic ferment-producing bacteria predominate in the infant's intestine (especially the obligatory milk-feces bacteria of Escherich and the large group of anaërobic butyric-acid bacilli). These energetic ferment-producing bacteria predominate very much over the genuine bacteria of putrefaction (especially the group of anaërobic putrefactive butyric-acid bacilli, bacillus putrificus coli; the spore-carrying type of gas-phlegmon bacillus, etc.). For this reason, acid fermentation prevails in the normal infant's intestines, and under physiological conditions (that is, when the child is nursed), putrefactive processes may be entirely prevented. The acid products of metabolism of intestinal bacteria have an exciting influence on intestinal peristalsis, and are essentially aided by the presence of intestinal gases.

Intestinal gases arise primarily from the life-activity of intestinal bacteria, and their constant presence in the intestine is probably not without importance. Intestinal gases play an important part in the topography of the abdominal contents, and regulate intestinal peristalsis. They keep the intestinal lumen open for the entrance of food, and the mingling of the latter with gases favors its transportation. The surface of the mucous membrane, without any doubt, becomes considerably greater from the distention of the intestines with gases. The villi are unfolded and the coiled vessels in the intestinal wall become dilated and stretched. This favors absorption and makes the circulation of the blood easier.

A very important rôle of normal intestinal bacteria is their ability to protect the intestine to a marked degree against the invasion of organized foes, through the products of their metabolism. The correctness of this view is demonstrated by our daily experience of the uniformity of the intestinal flora, as well as the fact that milk-feces and fecal culture-media check the growth of microbes that are foreign to the intestinal tract.

THE INTESTINAL FLORA OF THE INFANT UNDER PATHOLOGICAL CONDITIONS

Escherich has shown that a large number of intestinal diseases in infancy are associated with changes in the bacterial flora of the intestine, and was successful in discovering the specific causes of acute digestive disturbances in infants and in proving their etiological importance. The changes in the intestinal flora express themselves either in variations of those intestinal bacteria normally present in the field, or in the occurrence of new kinds of bacteria not originally indigenous to the intestine. The variations of intestinal bacteria within the normal field may limit themselves to the presence of a type of bacteria found only exceptionally in the normal picture, or may consist in an increase or a diminution in the normal number of intestinal bacteria.

Sometimes such alterations are brought about by a change in the diet (viz., artificial feeding), without any disturbance of the activity of digestion. Very frequently such a condition of the intestinal flora is associated with an abnormal condition of the feces and with pathologic changes in the intestines. The alterations of the bacteria are usually of secondary nature, and to be considered as the result or the expression of existing intestinal catarrh. The increased water content of the intestines favors the growth of certain kinds of bacteria; and increased peristalsis brings down the normal inhabitants of the upper sections of the intestine, which in normal conditions are not encountered under the microscope.

It is easy to see that changes in the bacterial flora of the intestine, with predominance of a chemically active type, may have a harmful influence upon intestinal digestion. The predisposition to it must, of

course, exist, in the shape of an abnormal composition of the food-remnant. So long as the combined action of intestinal bacteria and their relations to one another in the intestine or on a medium that can imitate the natural conditions with sufficient closeness, have scarcely been studied at all, we can give vent only to vague suppositions upon this topic, which have little value.

French investigators (Gilbert, Girod, Lesage, and Macaigne) have maintained the theory, from the study of the diarrhœal stools of infancy, that the normal bacillus coli may take on an increased virulence in the intestine under certain conditions, and so be the direct cause of intestinal disease. Escherich has opposed these views. Nevertheless, the fact is noteworthy that a great number of the microbes that have been encountered as the exciters of acute digestive disturbances in infancy (bacillus coli, streptococci, acidophile bacilli, and gas-phlegmon bacilli) show very marked similarities to a series of bacterial types that may ordinarily be obtained by culture from the feces of healthy breast-fed infants. However, there is much to indicate and prove that these are not identical forms, but different types from the various groups of bacteria.

That large group of acute intestinal diseases, which Escherich has designated ectogenous specific intestinal infections, is characterized by the presence of a new form of bacteria in the infant's intestine. The causal relation of the microbes found to the disease process has been determined by varied observations, and made likely by the following facts:—(1) the marked predominance of the bacteria in the microscopic field, which gives it, as a rule, its characteristic appearance; (2) the constant presence in considerable number, in the culture, of the forms of bacteria considered the responsible causes of the disease; (3) the penetration of the bacteria intra vitam through the damaged mucosa into the blood, the urine, and the various organs; (4) the contagious nature of the cases; and (5) the epidemic outbreak of similar disease processes in children's hospitals. The best evidence of the specific character of individual infections is a positive serum-reaction. The pathogenicity of the bacteria for laboratory-animals is here of subordinate importance.

The classical picture of an acute specific intestinal infection was given by Escherich in 1899, and was subsequently observed repeatedly in many clinics. This was the *streptococcus-enteritis* of infants. Escherich's description of this disease answers all the conditions that can be required to demonstrate a specific etiology. To be sure, we do not always have to do with one and the same species of streptococcus, but with different varieties from the large group of intestinal streptococci, which, as Escherich assumes, penetrate from the external world into the infant's intestine.

The diagnosis of specific streptococcus-infection is made most easily and plainly from a study of the preparations of feces stained after Weigert-Escherich. An illustration given of a sporadic case observed recently in the Vienna Clinic (Plate 46, Fig. V) shows sufficiently well the characteristic field and its remarkable variation from the normal condition; besides the large number of bacteria of the colon group present, the streptococcus completely dominates the field of vision. The colon bacilli have no share in the etiology of this disease, as agglutination-tests have shown. Culture of the streptococci from the feces is made most successfully by inoculating small particles of the intestinal secretions on grape-sugar bouillon, variously diluted. By this elective method, streptococci often show almost a pure culture in the last dilutions. The streptococci can then be isolated from this base by plate culture. In typical cases, they exceed in number all other bacterial colonies on agar plates directly smeared with fecal matter.

In the fall of 1898, Escherich observed a devastating epidemic at the clinic at Gratz. It took the form of severe vomiting and purging, and within a short time carried off a large number of infants. A very characteristic picture was given by the bacterioscopic investigation of the feces, which, superficially considered, resembled that of the normal feces of a breast-fed infant; and was characterized by the predominance of rods staining by Gram's method (Plate 46, Fig. VIII). All other forms of bacteria were subordinate to these. On this basis, Escherich designated the disease as "blue bacillosis." This name indicates, at the same time, that there were more than one type of bacteria present; in fact, quite a number of varieties, but all possessing the common characteristic of resisting decoloration with iodine-iodide of potassium solution.

In the majority of cases studied, Escherich identified a rod staining with Gram's method as the probable producer of the disease. These rods grow into long, curved threads, and show genuine branchings in the culture. The bacillus belongs in the group of acidophile bacteria, and has the greatest similarity to the bacillus acidophilis, which I have isolated and described in the normal stools of the breast-fed infant. Its isolation from the feces and culture are most successful on acid nutritive media. In the intestinal wall or sections from the organs of infants that had died during the epidemic, the bacilli could be repeatedly demonstrated. Finkelstein reported similar cases at the same time, from Heubner's clinic. In another series of cases in the same epidemic, Escherich isolated a short rod staining by Gram's method, which, in form and characteristics, most clearly resembled the Löffler-Hofmann pseudodiphtheria bacillus. Although morphologically similar bacilli were present within the intestinal wall, and could be grown in two cases from the spleen and kidneys, Escherich does not venture to lay down with certainty their etiological relation

to the attacks of gastro-enteritis. Whether anaërobic forms of bacteria have a part in causing the disease known as blue bacillosis has not been investigated. From our recent investigations, it seems probable that in some of these cases the anaërobic bacteria play an important rôle.

Escherich and Pfaundler have held that the bacillus coli communis is responsible for a third group of infectious intestinal inflammations in infants and older children. Finkelstein had previously recognized the same bacteria as the exciting factor of a hospital-epidemic of follicular enteritis occurring in Heubner's clinic. Escherich described this disease as *"Coli-colitis contagiosa."* It presented the essential symptoms of inflammation of the colon. Preparations of the feces show a typical bacterial picture, which is hard to differentiate from that of a coli-cystitis (urinary sediment) (Plate 46, Fig. IV). The bacillus coli communis is found in the stools in pure culture. The correctness of the view that the pathogenic types of coli enter the intestine as strangers from without is best proved by the eminently contagious character of this disease. Very remarkable and interesting is the determination of the fact that the pathogenic colon bacteria are agglutinated in quite marked dilutions (Pfaundler), in marked contrast to the autochthonous species of coli from the blood serum of the sick child; and the frequent occurrence of a coli-cystitis directly following the intestinal disease. The disease picture and the assertion of Escherich that the active agents of the disease isolated in these cases belong to different varieties of the colon group, of which some species cause fermentation and some do not, bring this type very near to that of acute dysentery. The etiological differentiation must, however, be maintained, corresponding with the results obtained by the serum reaction.

Shiga, Kruse, and Flexner discovered and described the agents of infection in bacillary dysentery —a disease that we know occurs much less often in infancy than in later childhood. Whereas the bacilli described by Shiga and Kruse were later proved to be identical, the bacillus Flexner does not belong to the group Shiga-Kruse; nor does the bacillus coli, with which it shows great external similarity. The bacillus Shiga-Kruse and the Flexner bacillus, in contrast to the bacillus coli, have no flagellæ; are immovable; do not ferment sugar; and do not coagulate milk. The bacillus Flexner forms acid on a culture-medium of mannite sugar, but the bacillus Shiga-Kruse does not. The differentiation of all three types is best and most rapidly made by the method of Jehle, as given.* The bacillus coli, by producing acid,

* Four parts of distilled water are mixed with one part of bovine serum, and to this mixture one per cent. mannite sugar (Merck) and one per cent. of a five per cent. litmus-solution are added. The sterilized mixture represents a clear bluish-colored fluid. One cu. cm. portion of this nutritive medium is poured into small test-tubes, and suspicious colonies from the plate-cultures of the stools are inoculated into the various tubes. This method has the advantage over Drigalski's in that it also gives us information on the formation of gas. Moreover, the smallest bubbles of gas become fixed at the same time by the agglutination of the albuminous substances; whereas, ordinarily, they easily break and escape observation.

colors the nutritive medium red and solidifies the column of fluid. To this column and its surface, small gas bubbles cling; and the nutritive medium appears torn apart by the formation of gas. The bacillus Flexner causes a similar reddening and coagulation of the nutrient medium, but the column remains homogeneous. There is no trace of gas bubble formation. The bacillus Shiga-Kruse leaves the nutritive medium blue and fluid. The most certain method of differentiation for all cases is the specific serum reaction.

Recently very thorough studies have been made in dysentery and dysenteric diseases (Hastings, Pease and Shaw, Wollstein, of America; Leiner, Jehle, of Vienna). In some epidemics the bacillus Shiga-Kruse, and in others the bacillus Flexner has been found to be the exciting factor. From all reports, the latter seems to be more commonly the cause of dysentery in childhood than the former, especially considering the cases that occur sporadically (Plate 46, Fig. VII). The bacilli of dysentery are completely absent from the stools of the normal infant, and have never been found in severe enteritis of the type known as cholera infantum (Jehle).

Case reports on intestinal infection with bacteria not included in these four prinicipal groups are of subordinate importance. I have been able to demonstrate the staphylococcus albus in large numbers in the stools of breast-fed infants suffering with acute intestinal catarrh. The marked predominance of these bacteria in preparations from the feces of the naturally-fed infant was very noticeable (Plate 46, Fig. III). "Staphylococcus-enteritis" is of especial interest, because it constitutes an infection peculiar to children at the breast. Kermauner has recently made similar observations in the Heidelberg Maternity Clinic, and has identified these bacteria as the causal factors giving rise to epidemics of infectious intestinal catarrh in breast-fed infants within that institution.

In connection with a small epidemic of pyocyaneus infection in the Gratz Children's Hospital, Escherich found the bacillus pyocyaneus in the diarrhœal stools of the diseased infants, and saw in the presence of this malignant pus producing organism in the intestine the cause of the local affection. Booker first drew attention to the importance of proteus vulgaris in the etiology of gastro-enteritis. Brudzinski subsequently investigated a great many stools for proteus, and very frequently demonstrated its presence in the foul smelling, compact, clayey evacuations of artificially fed infants. On Escherich's suggestion, Brudzinski carried out an interesting experiment. The proteus could be driven from the intestine by giving large amounts of milk-sugar, or by feeding directly with fresh cultures of bacillus lactis aërogenes; and the stools regained their natural acid odor.

While an important rôle in the etiology of acute digestive dis-

turbances should properly be ascribed to the anaërobic butyric acid bacilli, of which Klein has given us an interesting case (bacillus enteritidis sporogenes), the peptonizing bacteria of Flügge take only a subordinate position as the exciters of infectious intestinal diseases in infancy, and seem to have no directly specific action (Spiegelberg).

Description of the Bacterioscopic Pictures of Intestinal Flora.

Plate 46; magnified seven hundred times (Homog. immers., Winkel). Coloring according to Weigert-Escherich, No. 7, stained with methylene-blue. I. Preparation of meconium richly variegated in form, but poor in species. Spore carriers and free spores. On the left, somewhat below the middle of the field, a branched rod (bifidus). II. Preparation of a normal stool (cow's milk), rich in form and in species; predominance of bacteria negative to Gram's stain. Predominance of bacteria of the coli group. III. Preparation of the watery stool of a breast-fed child with acute intestinal catarrh. Picture of the so-called "staphylococcus-enteritis." The bacteria staining by Gram's method, which are so characteristic of the stools of the breast-fed child, are here in the background. The pyogenic staphylococci predominate. IV. Preparation of the mucopurulent portion of a stool from colitis. The so-called coli-colitis. Many pus cells. The bacillus coli in pure culture, partly intracellular. V. Preparation from the feces of an artificially-fed child with acute intestinal disease. The so-called "streptococcus enteritis." Mostly streptococci, sometimes arranged in long chains. Besides these, bacillus coli. VI. Preparation from the stools of a healthy breast-fed child. Uniform picture (bacillus bifidus). The simple branching and head carrying forms of bifidus are seen. VII. Preparation of a collection of pus from the feces of a case of dysentery. Many pus cells. Enclosed in them, numerous short rods. VIII. Preparation from the watery part of the stool of an artificially-fed infant with acute intestinal disease. The so-called "blue bacillosis." The bacteria staining by Gram's method predominate. Besides these, bacillus coli in small numbers. IX. Preparation of a colony of bacillus bifidus grown anaërobically on glycerin-sugar-agar. Numerous antler like branchings and bulbous poles.

POISONS

BY

PROFESSOR A. SCHLOSSMANN, OF DUSSELDORF

TRANSLATED BY

DR. CHARLES F. JUDSON, PHILADELPHIA, PA.

I. GENERAL CONSIDERATION

WE consider poisoning to be those phenomena and changes which are brought about by the action of poisons on the organism. What a poison is, is not easily defined since one and the same substance, according to the amount in which it is absorbed, and according to the condition in which it is taken into the organism, and also according to the condition of the body into which it enters, may have more or less toxic action. Poisoning is not then an absolute, but a relative, condition. Every substance which acts upon the constituents of the child's body may become toxic under certain conditions. The essential condition for the production of such toxic action is that the poison must be in solution or must become soluble.

We must distinguish ectogenous intoxications and endogenous intoxications (autointoxications). In the *endogenous intoxications* the substanc which acts as a poison is manufactured in the child's body. In *ectogenous intoxication* the poison is introduced into the body as such or in combination, from which it is freed and made soluble by the secretions of the body. The ectogenous poisons are those with which we have mainly to do.

Poisoning is not very rare in childhood. It is true that certain factors which play an important rôle in adult life seldom play a part in childhood, namely, suicide and the absorption of poisonous substances incidental to some trades. On the other hand the unreasonable habit of putting everything to the mouth, and the circumstance that even small amounts of poison can produce very severe effects in children are of great consequence in the causation of some poisonings. In prophylaxis we must especially consider these two last-mentioned factors. Every substance which can act as a poison or as a corrosive must be kept away from the child's environment and well out of reach to avoid accidents. In deciding on the dose of medicine we must always consider the age of the patient for whom the medicine is intended and regulate the amount of the various constituents, accordingly the bottle should have the exact prescription (do not write: "according to the physician's

directions"; instead, write "a teaspoonful every three hours", "6 drops 5 times a day," etc.). When prescribing dangerous remedies I always oblige the mother or nurse to repeat my directions and I am careful in giving opium, santonin, etc., to emphasize the injury which may result from over-doses. Even in prescribing medicines to be used externally care is necessary when these are poisonous. In prescribing baths of corrosive sublimate, for instance, I impress the mother strongly with the necessity of keeping the mercurial lozenges locked up that the other children may not be tempted by the sight of the red pills and put them into their mouths. One must also take pains that the patient in the tub does not swallow the solution and that none of it reaches the mouth, nose or ears. In prescribing poisons one should limit oneself to the minimum dose necessary. Unfortunately unused drugs are frequently kept and may subsequently fall into careless hands.

GENERAL TREATMENT OF POISONS

In the therapeutics of poisoning we must act rapidly, as we have little time to consider. He who gives at once, gives double measure. This applies especially in the dangerous conditions brought about by poisoning in childhood. We must endeavor to ascertain immediately with what poison we have to do to be able to improvise therapeutic remedies. To prevent evil results from poisoning we must attempt:

1.—To remove the poison from the body.

2.—To bring about the chemical transformation of the poison into a nonpoisonous substance.

3.—To give remedies which have the contrary physiological effect.

1. THE REMOVAL OF POISONS FROM THE BODY

Many poisons lead to reflex movements of the body by which they are partly rejected (spitting and vomiting). Our first attempt must be to remove all that is possible mechanically from the mouth and pharynx and to free the stomach from its contents. We must attempt to cause vomiting by irritation of the posterior wall of the pharynx and rapidly follow this with a thorough evacuation of the stomach by the tube. Under some circumstances lavage may also be required. We must take into consideration two facts: in some poisons (lye, acid, corrosive sublimate) we encounter at times deep ulceration of the œsophagus and gastric wall so that perforation by the tube is quite possible; therefore, a soft rubber tube must be used and great care taken. In washing out the stomach we must consider further that the poison must on no account be made soluble. In many cases, therefore, we should not use water for lavage, but in preferance milk or an albuminous solution (for example, after swallowing corrosive sublimate). In other cases milk is contraindicated on account of its fat content (phosphorous poison-

ing). Some poisons enter the circulation and are then thrown into the stomach with the gastric secretion and then absorbed again. Lavage must then be repeated, even if the patient is reached sometime after the drug has been taken (opium, morphine, iodine, etc.). Apomorphine hydrochloride may be used hypodermically to cause vomiting, 0.0008–0.0015 Gm. ($\frac{1}{75}$ to $\frac{1}{40}$ gr.) in children under 2 years of age, 0.002–0.005 Gm. ($\frac{1}{30}$ to $\frac{1}{12}$ gr.) in children from 2 to 10 years, over this age 0.01 Gm. ($\frac{1}{7}$ to $\frac{1}{4}$ gr.). To prepare the solution quickly we should have tablets of apomorphine 0.01 Gm. ($\frac{1}{6}$ gr.) ready, which can be dissolved in 1 to 5 c.c. (ɱ 16–1½ dr.) of water according to the dose which one wishes to give. Emetics which act on the stomach directly should not be given and even the use of apomorphine may be avoided by causing vomiting mechanically.

Whatever part of the poison has reached the intestine must be removed as rapidly as possible per rectum. Lavage of the intestines and high clysmata set up peristalsis in the upper parts of the intestine and help to evacuate the small intestine. (A pint to a quart of lukewarm salt solution must be introduced with the irrigator to which soap-suds may be added to increase the peristalsis.) If the poison has entered the body directly from the intestines (clysma of sublimate, carbolic acid, etc.) one should irrigate the bowel with milk or white of egg solution. The use of medicinal purges cannot be avoided at times, the saline purges are the best; for example, Carlsbad salts (1 to 3 teaspoonfuls to a tumbler of hot water). Castor oil is contraindicated in phosphorus and cantharides poisoning.

The skin and kidneys may also help to remove the poison from the body. To increase the secretion of the kidneys and sweat glands we can give large amounts of fluid especially in the shape of milk diluted with carbonic acid waters (one part of milk with three parts of Selters water or Vichy or Wildungen may be given). Where the condition of the stomach does not admit of giving water we must give repeated rectal injections and give normal salt solution subcutaneously in 0.9 to 1 per cent. solution (this hypertonic solution is desirable since it causes withdrawal of fluid from the tissues and thereby aids in elimination of the poison). Diuresis must be further excited by remedies stimulating the kidneys, for example, diuretin 1.0–3.0 Gm. (15–45 gr.), water up to 100 c.c. (3 oz.), 1 teaspoonful every one to three hours. Caffeine sodium benzoate, 1 Gm. (15 gr.), water up to 100 c.c. (3 oz.), one teaspoonful every one to three hours.

The subcutaneous injection of caffeine is of use (caffeine sodium salicylate 1 Gm. (15 gr.), water up to 10 c.c., inject 0.05 to 1 c.c.—1 c.c. =0.1 Gm. caffeine sodium salicylate=0.05 Gm. caffeine).

The action of diuretics on the kidneys can be heightened by stimulating the action of the heart (camphor, digitalis); this also stimulates

the secretion of the skin, which can be excited by hot packs, giving hot flaxseed tea, or sometimes by injections of pilocarpine (pilocarpine hydrochlorate, 0.05 Gm. aquæ ad 10 c.c., half a syringefull, 0.0025 Gm. ($\frac{1}{24}$ gr.) up to one whole syringe 0.005 Gm. ($\frac{1}{12}$ gr.) or two syringes 0.01 Gm. ($\frac{1}{6}$ gr.). We must make sure of the permeability of the kidneys before giving diuretics. Venesection followed by infusion of normal salt solution has proved of service when the poisons have entered the circulation and caused deep unconsciousness.

2. CHEMICAL TRANSFORMATIONS OF POISONS

We must endeavor to transform a soluble harmful substance into a soluble but non toxic one or to change the soluble poison into an insoluble combination. (Sulfuric acid poisoning—give sodium bicarbonate; lye poisoning, give lemon juice to unite the alkali; nitrate of silver—give table salt to form insoluble chloride of silver.) The action of antitoxin, producing immunization, is explained in a similar way.

3. THE USE OF DRUGS WHICH ARE PHYSIOLOGICAL ANTAGONISTS

We are familiar with many substances which have a direct or specific action on certain parts of the body, for example, exciting or paralyzing nerve cells, contracting or relaxing involuntary muscles. The physiological actions of these chemicals may be so intense as to endanger life. We must then set up a contrary reaction to overcome the first effect, for example in morphine poison inject atropine. Beside the direct combating of the action of drugs, careful attention must be given to the general health by rest in bed and prevention of all excitement, with suitable diet according to the peculiarities of the case; in acid poisoning the use of gruels (barley or oatmeal), in protracted vomiting ice cold nourishment

We must watch the breathing when the patients are unconscious and pay especial attention to keeping the tongue drawn forward or artificial respiration may be necessary. The supine position or elevation of the foot of the bed will prevent cerebral anæmia. Hot water cans or hot baths are necessary when the temperature of the body is low. The after-treatment is often long drawn out.

Under all circumstances our prognosis must be cautiously made since unexpected complications are not unusual.

II. SPECIAL POISONS

A. INORGANIC POISONS

(*a*) POISONING BY GASES

In childhood carbon monoxide poisoning is the only one frequently encountered. The *causes* are: escape of illuminating gas; the formation of coal gas containing from three-tenths to six-tenths per cent. carbon

monoxide by burning wood and coal, with insufficient entrance of air (too early closing of door of stove).

Symptoms.—Malaise, vertigo, vomiting, dyspnœa, coma and convulsions. Post-mortem findings: bright red spots and stripes, the typical appearance of the blood as seen with the spectroscope.

Treatment.—Plenty of fresh air, open window or carry the patient out of doors, rub the skin or give mustard bath. Inhalation of oxygen, stimulation such as camphor and ether injection, coffee, artificial breathing. The youngest case reported in literature concerned an infant twenty-nine days old who recovered in 11 days.

(*b*) POISONING WITH ACIDS AND LYES

Concentrated mineral acids and powerful lye, such as ammonia in the shape of sal ammoniac, are used for cleansing purposes in most households. The foolish habit of keeping such powerful caustic fluids in beer bottles, soda water bottles and the like often has unfortunate results.

Symptoms.—Ulceration of the parts reached by the fluid. Destruction of the epithelium, and ulceration of the deeper layers, erosion of blood vessels, complete perforation of the œsophagus or stomach. The fluid taken is in part expectorated, soon afterwards vomiting occurs with the rejection of epithelial tissue and masses of mucus, sometimes also blood.

Violent pains and restlessness and great weakness with weak pulse, at times symptoms of internal hæmorrhage. The further course varies. Recovery may ensue within a short time, or sudden death result in some cases weeks later as the result of gastric perforation. More frequently permanent and tedious injuries ensue in the shape of scar formation, especially in the œsophagus. Œsophageal strictures follow very frequently after lye poisoning and may be impermeable for fluid as well as solid nourishment.

Treatment.—Rapid removal of the poison from the stomach by lavage and neutralization of the poison; for acids, sodium bicarbonate, magnesia calcined 10 Gm. in 500 c.c. water, soapy water, chalk, albumin water, milk; for lye, vinegar, lemon juice, milk. The pain may be relieved by cocaine and glycerin, swallowing of ice pills and ice cold food. Consecutive stenosis of the œsophagus must be guarded against by prolonged use of the œsophageal sound. Gastrostomy, and dilatation of the stricture with shot.

(*c*) POISONING WITH HALOGENS AND OTHER COMBINATIONS

Symptoms.—Rapid loss of flesh, loss of appetite, great irirtability, skin eruptions. Bromide acne, coryza and œdema of the glottis from iodine. Examine urine.

Treatment.—Remove the drug. Give baths and stimulate the sweat glands. In the acute stage starchy decoctions and injections, mashed potato, sodium subsulphate 3 Gm. aquæ ad 100 c.c., in teaspoonful doses.

Potassium chlorate was formerly much used in diphtheria; it is still employed as a gargle and sometimes taken internally by mistake.

Symptoms.—Methæmoglobinuria, yellowish green to brownish color of the skin, vomiting, nephritis, uræmia. The urine is colored greenish brown and contains fragments of red blood corpuscles.

Treatment.—Washing out the stomach and intestines. Diuresis, give sodium bicarbonate, venesection.

(*d*) POISONING WITH METALS AND METALLIC COMPOUNDS

Mercury.—The most common form of mercurial poisoning observed is that with corrosive sublimate. Irrigation of the intestines or of the bladder with corrosive sublimate solutions is dangerous.

Symptoms.—Ulceration, salivation, vomiting, severe colicky pains, tenesmus and nephritis.

Treatment.—Gastric lavage with milk; lavage of the large bowel; plenty of water.

Lead.—Lead soldiers, or toy printing presses, the drinking of acetate of lead solution through mistake, cosmetics containing lead, nursing from the mother's nipple anointed with white lead.

Symptoms.—In chronic poisoning; anæmia, lead colic, paralysis, convulsions, blue line on the gums at edge of teeth; in acute poisoning, dyspepsia, pain in stomach, anæsthesia, convulsions, amaurosis; amblyopia occurs in chronic poisoning.

Treatment.—Sodium or magnesia sulphate, plenty of milk in acute cases, iodide of potash 4 grains 3 times a day. In the chronic cases the lead colic requires symptomatic treatment (opium).

(*e*) PHOSPHORUS AND ARSENIC

Phosphorus.—Only the yellow phosphorus is poisonous. Medicinal preparations which may cause poisoning are solutions of phosphorus in oil or with cod liver oil. The heads of matches containing phosphorus are frequently swallowed with suicidal intent. Rat poison often contains phosphorus.

Symptoms.—Acute gastric symptoms with vomiting of phosphorescent matter, smelling of garlic. After a few days enlargement of the liver, jaundice, diarrhœa. In the urine albumin and biliary coloring matter. At the autopsy advanced degeneration of the liver and heart, hæmorrhages from the stomach and mucous membrane. One should not confound acute cases of sepsis with phosphorus poisoning.

Treatment.—Milk, castor oil and fats must on no account be given. Wash stomach thoroughly with one-tenth per cent. to one-half per cent. solution of potassium permanganate. Give as emetic 0.1–0.5–1.0 Gm. (2 to 8 to 16 gr.) of sulphate of copper well diluted, and from time to time give teaspoonful doses of a solution of sulphate of copper, 1 Gm. (15 gr.) in one pint of water. Old oil of turpentine of acid reaction is a useful antidote; 1.0 Gm. (15 gr). may be given dissolved in a cup of oatmeal gruel and repeated.

Arsenic.—Accidental taking of the drug, criminal attempts, chewing and licking of toys, carpeting, etc., colored with arsenic (the latter a very rare form of poisoning at present) excretion of arsenic with the breast-milk. A man tries to poison his wife; the infant she nurses dies and arsenic is found in the cadaver.

Symptoms.—Vomiting, cholera-like diarrhœa, tenesmus, cramps in the extremities, convulsions.

Treatment.—Lavage of the stomach and intestines, hypodermoclysis. The freshly prepared antidote (hydrated oxide of iron) in tablespoonful doses every 10–15 minutes. Dilute burnt magnesia with 15 to 20 parts of water; give 2–3 tablespoonfuls every 10–15 minutes. Stimulants, hot pack, camphor injections.

B. ORGANIC COMPOUNDS OF CARBON

(*a*) ALCOHOL

Alcoholic intoxication of acute and chronic type occurs only too often in childhood. Acute alcoholism usually results from carelessness in leaving the whiskey bottle or flask within the child's reach; sometimes the parents or caretakers give excessive amounts of alcohol to their children. Chronic poisoning with alcohol usually follows the habitual use of beer or wine (especially sweet wine) which are given to the children to "tone them up" or because "the child should drink with us."

The usual symptoms of drunkenness (in acute poisoning) are rapidly succeeded by loss of consciousness, deep coma with pallor, cyanosis of the face, cold moist skin, and a scarcely palpable pulse which may be irregular. Convulsions and death may follow. Chronic poisoning results in poor physicial and mental development, neuropathic conditions, psychoses, delirium tremens, cirrhosis of the liver; the same phenomena are observed as in adult life.

Treatment.—Whatever one's views may be on the question of total abstinence, it is a fundamental principle of pediatrics that alcohol must not be given to children in any form or amount, even if only occasionally. The physician who yields to the parents importunities in this respect, fails in his duty as a professional man.

Alcoholic intoxication in children requires evacuation of the stomach, emetics or lavage, and washing out of the large bowel, followed by absolute rest, and maintenance of the temperature by artificial heat (hot-water bottles, etc.). Injections of caffeine sodium salicylate, and artificial respiration may be necessary. Ice cold applications to the head are grateful. Chronic poisoning must be treated symptomatically: alcohol must be absolutely withdrawn.

(*b*) METHANE DERIVATIVES

Chloroform.—Poisoning may result from chloroform anæsthesia: if the drug is given with due care (drop by drop) such accidents may easily be avoided, since the child's heart is usually strong and free from disease. The treatment of cardiac failure during narcosis consists of fresh air in abundance, pulling forward the tongue, artificial respiration, injections of camphor and electricity.

Iodoform.—Soon after the introduction of the drug for the local treatment of wounds many cases of poisoning were reported. Infants are especially susceptible.

Symptoms.—Skin eruptions, nausea, vomiting, restlessness, jactitation, psychical phenomena.

Treatment.—Remove iodoform from wound and dressings, excite the activity of the skin and kidneys.

Bromoform.—Deep coma, convulsions, loss of consciousness; treat by artificial respiration (and free stimulation.—Ed.).

Formaldehyde.—In gaseous form this substance irritates the mucous membranes; taken internally, even much diluted, it may give rise to necrosis of the mucosa.

(*c*) BENZOL DERIVATIVES

Carbolic acid.—Accidental taking of the drug; absorption from wounds, through the skin (carbolic acid sprays were formally much in vogue in the operating room.—Ed.).

Symptoms.—Pallor, vomiting, weak pulse, coma, convulsions, anuria. Test urine with iron.

Treatment.—Lavage of the stomach and large intestine, internally, give teaspoonful doses of a solution of caustic lime, 5.0 Gm. (75 gr.), in 40.0 c.c. ($1\frac{1}{3}$ oz.) of water, milk, magnesium sulphate and stimulants.

Acetanilid, antipyrin and lactophenin in too large doses or where the individual susceptibility is marked give rise to toxic symptoms of the most varied types. The new laxative purgen must be used with great caution.

Creolin and *lysol* act like carbolic acid; poisoning by these drugs requires the same treatment as carbolic acid poisoning.

Salol liberates carbolic acid, when it is split by the intestinal secretions. All these drugs readily enter the mother's milk, and may so

endanger the infant's life. (Do not use carbolized dressings for the nursing mother.)

C. PLANT POISONS

(*a*) POISONING WITH DRUGS IN COMMON USE

Opium, morphine, codeine.—The dose may be too large, the wrong medicine bottle used; decoctions of poppy are given by country people to quiet their children. Children tolerate very little opium.

Symptoms.—Sleep, coma, convulsions, vomiting, contracted pupil, small slow pulse, cool skin, fall of body temperature.

Treatment.—Repeated washing out of the stomach, internally, tannic acid in solution. Atropine (hypodermically $\frac{1}{600}$ to $\frac{1}{120}$ grain) is the physiological antagonist of opium; or small doses of the extract of belladonna may be given internally. Powerful excitants are required.

Santonin (chenopodium), so frequently employed as an anthelmintic, causes toxic symptoms in too large or too frequent doses.

Symptoms.—Vomiting, headache, pallor, yellow vision, amaurosis, convulsions; the urine is colored yellow, becomes red when caustic soda is added.

Treatment.—Evacuation of stomach and intestines. Stimulate the heart. Control convulsions by chloroform or chloral.

Filix mas, used to dislodge tapeworms, in poisonous dose causes severe pain, convulsions, dimness of vision. Treat like santonin poisoning. Do not give fat or oils.

(*b*) PLANT POISONS

The *hemlock* (conium maculatum) plant resembles parsley somewhat; the roots, stems, and leaves are poisonous (especially in May) but the seeds are most toxic.

Symptoms.—Choleriform diarrhœa, convulsions and paralysis.

Treatment.—Evacuate the gastro-intestinal tract. Lavage and saline injections. Stimulants.

The *deadly nightshade* (solanaceæ) has marked toxic properties, due to the alkaloids atropine, hyoscyamine, scopolamine, etc., which it contains. The blossoms and berries of the belladonna plant, which resemble ordinary cherries and contain atropine, the black henbane (hyoscyanum niger) and the thorn apple (datura stramonium) belong to this group.

The **symptoms** of poisoning with the plants mentioned appear very suddenly, especially in atropine poisoning. Marked dilation of the pupil makes its recognition easy. The marked rapidity of the pulse, the difficulty in swallowing, the loss of consciousness with delirium and convulsions complete the picture.

Treatment.—Removal of the poison from the body. Morphine and pilocarpine are antidotes.

The *tobacco* plant also belongs to the group of solanaceæ; acute nicotine poisoning most frequently follows the first use of tobacco by children (generally in the form of cigarettes, cigars or pipe). Tobacco poisoning should be treated by emetics, milk diet and in severe cases by injections of morphine.

The *foxglove* (digitalis purpurea) on account of its beautiful color, tempts children to taste it. Poisoning may also follow the continued use of the drug in too large doses. The most prominent symptoms are irregular action of the heart and the dyspnœa which results.

The **treatment** must be symptomatic; we must always remember that preparations of digitalis may have a cumulative action.

The *colchicum* plant contains colchicine which can enter the milk of nursing animals and so lead indirectly to poisoning. The symptoms are those of gastro-enteritis—eventually convulsions set in.

Treatment.—Besides the removal of the drug the inflammation of the intestinal tract must be combated by opiates and suitable diet.

(*c*) POISONOUS MUSHROOMS

When mushrooms are plentiful, poisoning occurs not infrequently during the summer months from eating those varieties which are poisonous. These are the *Amanita bulbosa* (the false mushroom), which gives rise to cholera-like diarrhœa, with general cyanosis and meningitic symptoms. *Helvella esculenta* and *Morchella esculenta*, the poison of which is soluble in water (Symptoms: vomiting, abdominal pain, jaundice, restlessness, etc.) and the *Amanita muscaris* (toadstools) which gives rise to marked nervous symptoms, delirium, convulsions, and coma.

Treatment.—In every case we must remove all remnants of the poison as rapidly as possible. The inflammation of the gastro-intestinal tract must be treated symptomatically (cracked ice, hot compresses to the abdomen, restricted diet). Strychnine hypodermically will be of service in poisoning with toadstools. Other forms of mushrooms (*Boletus satanus*), also agaric, and milter, cause toxic symptoms in rare instances.

Ergotism results from the continued use of meal made from grain contaminated with the *Claviceps purpurea* or ergot fungus. In acute cases we have severe gastro-intestinal symptoms, spasms and marked cardiac symptoms which may cause death. In the chronic cases tingling sensations are felt in the hands and feet, which have led to the name "tingling disease," and may terminate in gangrene.

Treatment consists in the rapid evacuation of the drug, the administration of tannic acid and of salicylates. Opium is required for the treatment of chronic cases. *Lathyrism* and *pellagra* have their own literature, to which the reader is referred.

(*e*) SNAKE AND FOOD POISONING

In German territory the only poisonous snakes are the striped adder and the viper (vipera Redii). Snake wounds must be thoroughly washed, allowed to bleed, and cauterized with pure carbolic or chromic acid, chloride of lime solution or the hot iron. When poisonous symptoms arise, diuretics and heart stimulants must be used. The danger from the bite of the adder has been exaggerated.

Sausage poisoning (botulism) gives rise to gastro-intestinal symptoms followed by paralyses, often bulbar in type. At present our treatment must be symptomatic. In the future results may be expected from a specific antitoxic therapy.

Meat poisoning, oyster poisoning, and milk poisoning result from the formation of toxic substances out of albumin, due to the action of micro-organisms. The symptoms are like those of sausage poisoning, and require the same treatment.

DISEASES OF THE NOSE, TRACHEA, BRONCHI, LUNGS AND PLEURA

BY

Dr. E. FEER, Basle, Switzerland

TRANSLATED BY

Dr. THEODORE J. ELTERICH, Pittsburg, Pa.

ANATOMICAL AND PHYSIOLOGICAL PECULIARITIES OF THE INFANT

Anatomy.—The nasal passages and the nasopharyngeal space are very narrow in the newborn and in nurslings. The nasal cavity is shallow corresponding to the low facial part of the skull, so that even in slight tumefactions of the nasal mucous membrane, the entrance of air is interfered with or even prevented. The contiguous nasal cavities are scarcely marked in the newborn (that of the superior maxillary bone being still absent), and develop only with age according to the standard of development of the facial part of the skull. The shape of the infantile thorax deviates materially from that of the adult. This is shown most distinctively in the newborn and younger nurslings. While the thorax of the adult resembles an ovoid flattened postero-anteriorly with its wider portion directed downwards, that of the newborn is obtusely conical, expanding uniformly towards its base. The antero-posterior diameter is as long or almost as long as the transverse diameter, so that the chest appears well arched in front, while the lateral portions are flattened. The upper thoracic aperture is not well inclined towards the front as in older children and adults, but extends in an almost horizontal direction. Correspondingly, the breast bone is higher by about a body and a half of a vertebra in its position to the vertebral column. The course of the ribs, in an almost horizontal direction, and the obtuse epigastric angle of the lower thoracic aperture, are also characteristic. Owing to these characteristics, the thorax acquires its short appearance and assumes to a certain extent a posture of permanent inspiration. In contrast to the adult, the transverse processes, as well as the ribs as far as the angulus, actually leave the vertebræ in a straight transverse direction. The ribs are soft and pliable, the thoracic muscles weak. These conditions gradually change with the growth, and lead to the permanent shape of the thorax which is already manifest at the fourth or fifth year of age. The ribs grow out from the transverse processes to the rear and

then sharply to the side, so that the thorax becomes more wide than deep, making room posteriorly and laterally for the growth of the lungs.

At the same time the upper thoracic aperture with the sternum and all the ribs descend, bringing about the broader and larger thorax with the anteriorly inclined ribs of later life.

At birth, the circumference of the chest of the newborn is about 32–33 cm. measured at the level of the nipples. The increase in the first year is about 12 cm., in the second 3–4 cm., from the third to the seventh year 1–2 cm. for each year, from the eighth to the twelfth year 1 cm. for each year, towards adolescence (thirteenth to the fifteenth year) 3–5 cm. for each year. Accordingly, the chest circumference at the age of five years would be 52–53 cm., at ten years 61–62 cm. In the well developed newborn the difference between the chest circumference and half of the body length is 8–10 cm. in favor of the chest circumference. The smaller this difference the lower the viability, which becomes questionable as soon as this difference falls below 7 cm. The chest circumference, in vigorous infants, should exceed the circumference of the head at the latest with the third year, and with the fifteenth year it amounts to one half of the body length.

From the fourth to the fifth year the right side of the chest exceeds that of the left, the difference in older males amounting to $1\frac{1}{2}$ cm. The lungs of the newborn begin to expand with the first inspiration; frequently, however, several days are required for their complete expansion. They are at first very small (see Fig. 67); and being confined in the short and narrow thorax, their space is still more encroached upon by the large heart and thymus. With the expansion of the thorax, the lungs develop rapidly during the first months, but still remain relatively small during childhood. The proportionate size of the lungs compared with the heart, is in the newborn 3.5–4 : 1, at puberty 7.3 : 1.

At birth, the lungs weigh 60 Gm. ($1\frac{7}{8}$ oz.), at one year 140 Gm. ($4\frac{3}{8}$ oz.), at seven years 300 Gm. ($9\frac{3}{8}$ oz.). Their growth depends principally upon the increase in the breadth of the thorax. The lobes of the lungs bear the same relationship as in the adult. As the lungs and diaphragm gradually descend the lungs increase in length, although their lower border remains at the same level with the ribs, coinciding with the adult (Sahli). At the same time with the descent of the thorax and lungs opposite the vertebral column, there also occurs a lowering of the larynx and the trachea, so that in the newborn the bifurcation of the trachea is opposite the third dorsal vertebra and in the adult opposite the fifth (Mehnert).

From the foregoing, it may be readily understood, that the arched diaphragm is only on a slightly higher level (estimated according to ribs) than in the adult, but that it is less sloping in its peripheral portions.

Physiology.—The frequency of respiration during rest and sleep may be considered as averaging about:

Newborn	End of the 1st year	2nd year	5th year	8–10th year
40–45	25	24	20	18

respirations per minute.

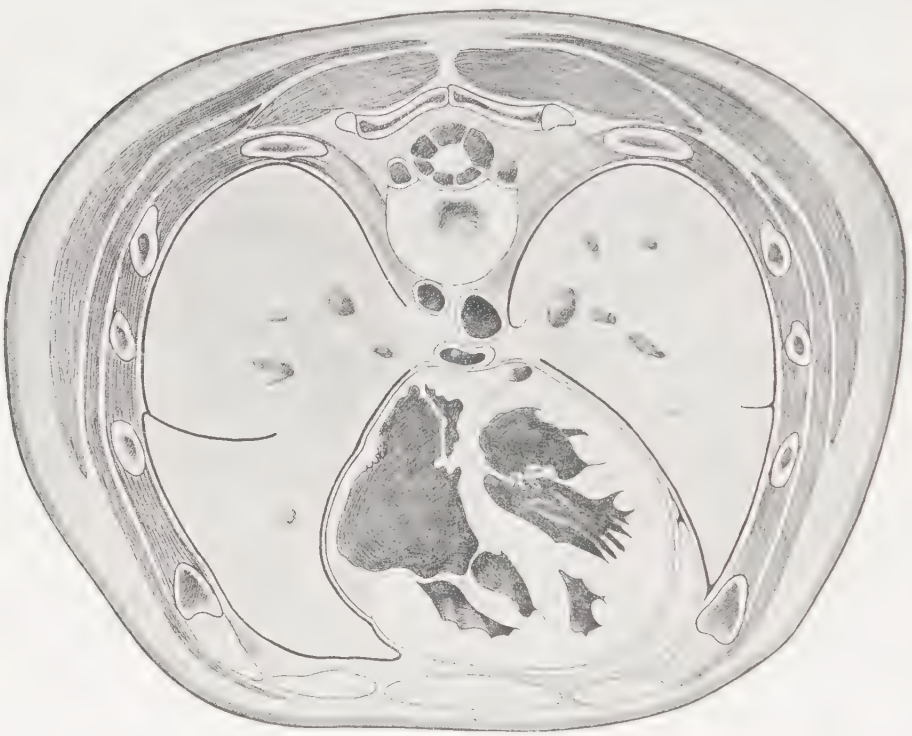
The respiratory frequency is lowest during sleep, is increased on sitting up, especially by excitement and crying (10–30 respirations), and is decreased by concentrated attention. It is, therefore, of great advantage, if the respiration can be observed during sleep, the frequency being determined by lightly placing the hand upon the chest.

During the early months and up until the third year, the respiratory rhythm is not always regular, even under normal conditions. This is especially seen in young nurslings during sleep when longer pauses may also occur (Czerny). In older children irregularities of respiration are usually of pathological significance. Infants frequently hold their breath for a long time on auscultation, which is always a proof that no serious disease of the respiratory system is present. During the first years the type of respiration is predominantly diaphragmatic (abdominal), and only at about the seventh year it is reinforced by an increased participation of the thorax. The type of respiration peculiar to sex begins to manifest itself at about the tenth year, the abdominal and inferior costal type being found in males and the superior costal type in females. During the early months of life an inspiratory recession of the epigastrium is physiological, but later points to disease in the respiratory organs, or to rachitis of the thorax.

We are indebted to the splendid investigations of Gregor for a more accurate knowledge of the mechanism of the infantile respiration. In young nurslings the respiration is diaphragmatic, frequent, and superficial. An increased demand for oxygen is supplied by an increased frequency of respiration, to double the usual number. The recumbent posture in which the weight of the intestines presses upon the lungs renders respiration more difficult. In the second half year and in the second year the capacity for frequent respiration is diminished, and the inspirations become deeper. (Heretofore, the respiratory movement took place almost entirely from its frontal axis; now, however, owing to the broadening of the sides of the chest, and the lowering of the ribs by the completed development of their necks, thus changing the axis direction backwards, a far greater respiratory capacity is permitted, in addition to elevation of the ribs outwards.) The ability to respire deeply is increased from the third to the seventh year, thereby causing an increased capacity of aëration. The frequency of respiration is greatly reduced from the eighth to the fourteenth year, and a diminution of the working capacity is brought about by progressive deepening of the respiration.

Gregor explains very satisfactorily the changes in the mechanism of respiration during infancy. As long as the infant is recumbent the respiration is solely diaphragmatic. Only by raising the body to the upright position, thereby initiating the descent of the anterior chest wall with the thoracic and abdominal organs, the increased thoracic respiration is brought about. As a matter of fact, the commencement of holding the body erect coincides with the blending of the diaphragmatic and thoracic respiration. According to Gregor, two factors are particularly

FIG. 67.



Transverse section of the thorax, at the level of the 5th costal cartilage of an infant 36 hours old (according to Mettenheimer). The wall of the right ventricle is of equal thickness with the left.

important for normal respiration: the great reduction of the frequency together with deepening of the respiration at the close of the nursing period, and the tendency to a reduction of the working capacity by reason of the acquired freedom of action* of the depth and volume of respiration after the seventh year. In infants having a tendency to frequent attacks of catarrhal bronchitis, Gregor observed a retarded development of the deep respiratory movements with the associated formation of a rigid and badly poised thorax.

The volume of air of each respiration (at 40–60 respirations during

*Freedom of action—Difference between the greatest and the minimum volume of respiration.

sleep) in the first half year is 27–42 c.c., in the second half year 78 c.c., and towards the end of the first year 136 c.c. (Gregor).

The absolute respiratory volume (expired air in one minute) increases from 2500 c.c. in the middle of the first year to 5000 c.c. in the eleventh and twelfth year, and does not, therefore, increase correspondingly with the body weight.

The relative respiratory volume (expired air per kilo and minute), amounts to 330–500 c.c. in the newborn, 563–533 c.c. in the seventh month, and at the end of the second year to 424–328 c.c.

The Chemistry of Respiration.—The insensible perspiration amounts, at first, to about 90 Gm. (3 oz.), at three months to about 200 Gm. ($6\frac{1}{4}$ oz.), at six months to 300 Gm. ($9\frac{3}{8}$ oz.), at one year to 500 Gm. (15 oz.) (Camerer). The excretion of water through the skin and the lungs, calculated according to body weight, is rather higher in the nursing infant than in the adult.

Comparing equal surfaces, the healthy breast-fed infant excretes rather less carbon dioxide than the adult, on the average 113 Gm. carbon dioxide per day; proportionately to the body surface 13.5 Gm. per hour.

Compared with the breast-fed baby, elimination of carbon dioxide is greater in the bottle baby (17.2 Gm.), partly owing to the greater activity resulting from a large supply of nitrogen with the cow's milk. In comparison with the body weight, infants absorb more oxygen and eliminate more carbon dioxide than adults, less, however, in comparison with the body surface (Rubner, Heubner, Bendix).

DISEASES OF THE RESPIRATORY SYSTEM

The diseases of the organs of respiration in the infant, may be considered the most frequent and important affections, and are only surpassed in importance, during the nursing period, by the gastro-intestinal disorders. As early as the second half year of life, the disorders of the respiratory system increase very rapidly; indeed, they occupy the first place in regard to frequency and mortality until the third and fourth year, occurring either primarily or secondarily to certain infectious diseases (whooping-cough, measles, influenza).

Krieger has shown in his splendid etiological studies (Strassburg, 1877) that after the beginning of extra-uterine life, the tendency to catarrhal inflammations and to other diseases of the organs of respiration, develops only gradually, passing downwards from the nose to the lungs. A coryza may occur in the newborn after the first week; but only later, at about the middle of the second month, the larger air-passages show a tendency to catarrhal inflammations, and as a general rule, still later, the finer bronchi and the lungs. The infant is born with healthy mucous membranes, and the noxious principles entering the body through the respired air require a certain length of time in order to render the

tissues susceptible to disease. Naturally, the periphery of the mucous membrane (the nose) is the first to be involved, since it is primarily exposed. The bronchi and lungs remain uninvolved, and are capable of resistance for a longer period, because they are more distantly situated from the outer world. The air passing through the bronchi is rendered harmless, or at least less harmful, by the known means of protection of the more peripherally situated mucous membranes (ciliated epithelium, bactericidal properties).

Moreover, the deeper seated a morbid process is, the later will the disposition to disease be established. For this reason, therefore, inflammatory conditions of the bronchi, rather than those of the lung tissue, occur during the early years of life.

DISEASES OF THE NOSE

Congenital Anomalies.—Congenital narrowing and complete occlusion of the nares are not so infrequently observed. The malformation, whether unilateral or bilateral, may involve the external nasal orifices, the nasal passages, and especially the posterior nares. Besides these, there are frequently found other malformations, which distinguish them from congenital adenoid growths of the pharynx. The obstruction in the posterior nares is, in most instances, osseous, more rarely membranous. In bilateral closure of the nares, a rapidly fatal asphyxia may occur after birth, since the newborn infant does not know how to breathe through the mouth, or a severe form of inanition may result in consequence of interference with nursing. Severe asphyxia in the newborn after birth, with impaired breathing and aspiration of the cheeks, with closed mouth, should lead us to think of this condition. If on opening the mouth and retracting the tongue, respiration becomes established, the obstruction is in the nose or nasopharynx. Examination by means of the air-douche and probe determines the site and nature of the narrowing or occlusion, respectively.

Whenever the anomaly causes severe asphyxia an attempt should be made at first to pierce the obstruction with a strong sound or trocar and to dilate gradually. In unilateral or partial closure the interference with respiration is often not of a serious nature, and only later leads to the need of medical advice. Improvement may take place spontaneously.

Deflections of the nasal septum frequently develop in children, in part as the result of trauma, from which also the external nose may suffer, in part spontaneously, in the course of years, from asymmetrical development of the bones at the base of the skull and the upper jaw. The deformity usually involves the anterior and inferior portion of the septum, and consists in a slight or considerable bending or crooking to the left or right with a horizontal axis. The diagnosis is readily made.

Only considerable curvatures and hypertrophies lead to interference with respiration, imperfect chest development, and even faulty formation of the orbits with astigmatism, etc. In such cases, in older children, a resection of the septum is to be taken into consideration, or eventually the removal of an inferior turbinated bone. In the milder cases, which are the most numerous, no treatment is required.

ACUTE RHINITIS (CORYZA)

Etiology.—Coryza attacks children more frequently than adults and, with especial predilection, nurslings. Mechanical, chemical (iodine), and thermal irritants often furnish the cause. Mostly, however, a coryza is infectious in its origin. Among the multitude of pathogenic germs (pneumo-, strepto-, staphylococci, meningococcus intracellularis), which are present, it is often impossible to determine which are causative and which are merely present as incidental attendants. Not infrequently, a seemingly simple coryza depends upon infection with true diphtheria bacilli, which quite often appear as a harmless accompaniment of the ordinary coryza in the nursling (Stooss, Ballin, Schaps), but only exceptionally upon gonococci. In infectious diseases coryza very frequently occurs secondarily; as a regular forerunner it is seen in measles, grippe, and influenza. The bacterial nature of a coryza explains without further comment why contagion is of such extraordinary frequency. According to my observations, the disease may also manifest itself in other members of the family as an angina or bronchitis.

Exposure to cold, no doubt, plays an important rôle in the production of coryza, and in young infants especially it may be held responsible with the greatest certainty (careless bathing, taking out in rough weather). The healthy nasal mucous membrane rapidly destroys the majority of the invading germs and harbors only a small number of them in its anterior portion. The beginning cold probably acts in such manner as to temporarily suspend the bactericidal properties of the nasal mucous membrane, so that the germs which are present, and those recently introduced by the respired air, may develop undisturbed. Personal predisposition is of great significance, and may be of a general or local character. Children suffering from chronic rhinitis and adenoid growths are repeatedly attacked by fresh colds from the most trivial causes; likewise also, anæmic, debilitated individuals. Frequent changes of temperature, cold east winds, unclean, dusty, but especially overheated dry air of the room, act injuriously. I have frequently observed hay-fever in children of four years and upward.

Symptoms and Course.—Redness and swelling of the nasal mucous membrane with copious discharge which, at first, is thin and watery, but later mucopurulent are the well-known symptoms of a simple catarrhal rhinitis. In severe infections (diphtheria, scarlet fever) the dis-

charge is often purulent (*rhinitis purulenta*). In older children, as in adults, coryza usually runs a harmless course and rarely leads to serious disturbances. On the other hand, in very young infants, particularly during the early months of life, it may become exceedingly menacing. In these cases, the disease at times commences with an elevation of temperature as high as 40 °C. (104 °F.). or even convulsions, and may seriously disturb the nutrition. In consequence of the very narrow nasal passages a complete stoppage of the nasal breathing easily occurs, which may lead to a dangerous asphyxia in the newborn, since they do not know how to breathe through the mouth. Cases are also recorded where the increased fruitless respiratory movements have caused a backward aspiration of the tongue; and threatened suffocation was only averted by drawing it forwards. The impeded or interrupted nasal breathing also constitutes an impediment to nursing, and every few moments the tortured infant releases the nipple in order again laboriously to draw some air into its lungs through the mouth. Sleep is also seriously disturbed, since the infant seeks to relieve its want of air by breathing through nose and mouth alternately. The laborious and overworked breathing causes great fatigue, suffocative attacks, and may lead to pulmonary atelectasis, which together with the inanition resulting from the insufficient ingestion of food, may bring about a fatal termination in the exhausted infant. Even sudden deaths have been recorded in severe coryza of the newborn (Baginsky) (without bronchiolitis?). In older nurslings also, as well as those of older years, coryza is attended with manifold dangers. At this age, the catarrhal inflammation is apt to extend, and often leads to false croup, bronchitis, and bronchopneumonia. The nasopharyngeal space and the tonsils quite regularly participate in the catarrh. Sometimes also, the coryza results in an enlarged tonsil. From thence, the inflammation is apt to extend to the Eustachian tubes and the tympanum. Otitis media is an exceedingly frequent complication of coryza in young infants. It may frequently progress unobserved, but may often cause high fever and convulsions, and may lead to rupture of the tympanic membrane, also to mastoiditis. Otitis media is especially favored by adenoid growths.

By the term *rhinitis fibrinosa* or *pseudomembranacea* is designated an affection of the nose which appears as an ordinary coryza with scanty mucopurulent discharge, but in which fibrinous membranes are expelled in a surprising manner, a process which may be repeated from time to time. Very often only one nostril is affected, so that the obstruction is not very great. Withal, the general condition remains undisturbed, and fever is absent, or there may be at the most a subfebrile temperature. The submaxillary glands are not enlarged, or only slightly so. The pharynx remains free, or a slight catarrhal condition may be present. The disease often is subacute or chronic, entirely be-

nign, and is not infrequently seen in older children. Formerly, the disease was regarded as entirely harmless, strepto- and staphylococci being found in the discharge. Later, however, in some cases, virulent diphtheria bacilli were demonstrated to be present in the discharge and in the membranes. It is quite evident, therefore, that it is frequently only an attenuated form of diphtheria, against which one must always be on guard since serious infections may follow in its wake (Gerber and Podack). In private practice it is always proper to regard rhinitis fibrinosa as diphtheria, as long as a careful examination has not demonstrated the absence of diphtheria bacilli. The treatment consists in the administration of antidiphtheria serum and mild irrigations. Insufflation of iodol is also recommended.

Diagnosis.—The diagnosis is usually readily made and does not require the use of a speculum. It is sometimes difficult at first to decide whether the appearance of a rhinitis is primary or whether it is the prodromal symptom of an infectious disease (influenza, whooping-cough, measles). In cases where the discharge is unilateral, purulent and bloody, a foreign body is usually present. A very purulent rhinitis in the newborn may be of a gonorrhœal nature. Of the highest importance is the differentiation of a simple rhinitis from nasal diphtheria, on the timely recognition of which the life of the patient often depends. Nasal diphtheria usually begins as a violent coryza and, even after careful examination with a speculum, often no formation of membrane can be distinguished, which is usually found in the posterior part of the nose in the direction of the nares. One must bear in mind that primary nasal diphtheria is comparatively frequent in young children, and that diphtheria is particularly apt to appear in this form during the early months of life. *Every case of rhinitis attended by fever, excessive purulent discharge, and markedly disturbed general health, is suspicious of diphtheria, and requires a bacterial examination.*

In cases of serious illness and well-founded suspicions, a serum injection should be made without awaiting the result of the bacteriological examination, which, owing to the almost invariable presence of the pseudodiphtheria bacilli, is not always promptly disposed of. Rhinitis pseudomembranacea progresses in the same manner, but much more harmlessly than nasal diphtheria.

The **prognosis** in simple rhinitis is good. Only in the newborn is there a direct menace to life; but older infants may also perish from a subsequent otitis and bronchitis, especially those suffering from malnutrition with disturbed digestion.

Prophylaxis consists in judicious hygiene and judicious methods of hardening. Association with persons afflicted with catarrh and sore throat should be avoided, likewise the use in common of the pocket handkerchief.

Special treatment of coryza is necessary only during infancy. Nasal

douches and injections are to be strictly avoided, since they are irritating and may easily cause an otitis media. In older children it is sufficient to remove all external injurious matters and to refrain from violent blowing of the nose. The free volatilization of the oil of turpentine in the vicinity of the patient is advantageous. An ointment of lanolin (bryolin, etc.) is the best protection against excoriation of the nasal orifices and the upper lip from the discharge. Infants suffering from a severe coryza should be kept in the room and, if feverish, in bed. Care should be taken to have the air of the room sufficiently moist during the season when the rooms are heated. During the acute stage considerable precaution must be exercised regarding the bath (a warm room, etc.). This had better be omitted in cases where the nursing is unreliable. Frequently, the discharge or dried crusts may be removed by means of a pledget of absorbent cotton, while the sneezing thus produced often promotes the discharge of the secretion. The instillation of a few drops of lukewarm and sterilized olive oil containing 1–2 per cent. menthol is of benefit, also an aqueous solution of cocaine 1–2 per cent., of which 1–2 drops are instilled several times daily in each nostril with the head resting obliquely. Lately, Ballin advocates the introduction of pledgets of cotton saturated with a 1: 1000 solution of adrenalin for 2–3 minutes several times a day. Ten to twenty drops of oleum pini pumilionis dropped on a piece of flannel, close to the head of the patient will often act beneficially. In cases of increased difficulty of swallowing, nourishment may be given with a spoon, and in emergency by means of the feeding tube. In a desperate case of a four weeks old infant, Heineman successfully performed tracheotomy. Older children may be allowed to inhale or be insufflated with small quantities (as much as will go on the point of a penknife) of finely powdered boric acid or borax containing 2 per cent. cocaine.

Hæmatoma and *perichondritis* are mostly frequently seen during childhood, the cause mostly depending upon a blow or a fall on the nose. A hæmatoma of the cartilaginous portion of the septum results, and this strips the perichondrium from the cartilage which is often fissured or necrotic. Usually, the hæmatoma becomes infected and leads to perichondritis and hypertrophy of the perichondrium and the mucosa. Interference with respiration usually occurs several days after the injury, and this leads the patient to seek medical advice.

On raising the tip of the nose a *dark red, smooth swelling* is at once seen on the anterior portion of the septum. It is usually bilateral, easily compressible with a probe, and not very painful. Since the septum is usually necrotic or perforated at some point the fluid contents of the swelling may frequently be moved about with the finger. Often the abscess ruptures spontaneously, or ultimately it may be emptied by a deep incision into the mucous membrane previously cocainized.

CHRONIC RHINITIS

Chronic rhinitis is characterized, anatomically and pathologically, by a marked cushion-like swelling and redness of the mucous membrane, most commonly seen in the region of the inferior turbinated bone with considerable enlargement of the venous network. A genuine hyperplasia of the tissue is less frequently present than one would expect from the great thickening. In prolonged cases, single portions of the mucous membrane may undergo atrophy.

Etiology.—Chronic rhinitis often develops from frequent recurrences of acute catarrhs, or it may develop gradually from the beginning. Occasionally it is found in connection with acute infectious diseases, especially after measles, and in isolated cases as the result of foreign bodies. The majority of cases are dependent upon a scrofulous condition, and are frequently associated with previously existing catarrhal conditions of the mucous membrane, eczema, and chronic glandular enlargements. With exceptional frequency, chronic rhinitis develops either concomitantly with, or as the result of, a hyperplasia of the lymphatic tissue of the pharyngeal ring. The disease is regularly found in connection with hereditary syphilis, these infants manifesting the well known symptoms of sneezing and snuffling. Later, in the course of the disease, there are added destructive ulcerations of the mucous membrane and the bony framework of the nose, leading to the well known and dreaded saddle nose. Uncleanliness and unsanitary habitations contribute largely to the production of chronic rhinitis and to the prevention of its cure.

Symptoms.—The increased secretion with its sequelæ is first in the line of symptoms in chronic rhinitis. The discharge is mucopurulent, often greenish in color, rarely slightly bloody, and not offensive. It contains bacteria, the strepto- and staphylococci predominating. The nose is constantly surcharged with mucus, and often requires several handkerchiefs daily to keep clean. The liquid secretion usually gathers in the lower nasal passage, while further above dried crusts are found. In addition, there is a copious discharge of secretion from the nose, causing swelling and excoriation of the nasal entrance and of the upper lip. The skin in the neighborhood of the nose often becomes eczematous. The secretion also flows backwards through the posterior nares into the pharynx, causing frequent desire to clear the throat, attacks of choking, swallowing and a teasing cough which often disturbs sleep. The increased difficulty of breathing through the nose causes the infants to keep the mouth open especially at night, and to snore during sleep. At night, the breathing is noisy when the mouth is kept closed. Pavor nocturnus is a frequent result. Changes for the worse and improvement frequently alternate. The colder season of the year with

the injurious atmosphere of the room regularly increases the trouble, as well as intercurrent infectious diseases. It is often difficult to decide to what extent chronic rhinitis participates in the obstruction of the nose, on the one hand, and how much may be attributed, on the other hand, to frequently coëxisting adenoid growths in the nasopharyngeal space. The adenoids are usually of greater moment.

The frequent mouth-breathing often produces catarrh of the pharynx and lungs. The insufficient pulmonary aëration leads to imperfect development of the chest (flatness); and an insufficient blood formation, frontal pressure, vertigo, migraine, and asthma are conditions often found in connection with chronic rhinitis. The voice acquires a nasal intonation. As complications, catarrhal and purulent otitis media and chronic conjunctivitis are first in importance. The diseased conditions of the accessory cavities, which play such an important rôle in the chronic rhinitis of adults, need not at all be considered in young children during the first years of life, because these cavities are very small and do not begin to participate until towards puberty. Abscesses of the antrum of Highmore, which perhaps may sometimes occur in younger children, are usually extensions of tuberculous processes from the alveolar process.

CHRONIC ATROPHIC RHINITIS (OZÆNA)

Formerly, all nasal affections having an offensive discharge were designated by the term ozæna. In recent times, the term has become restricted, and a number of affections whch are attended by ulcerations of the mucous membranes and the osteocartilaginous framework have been separated. To this class belong the syphilitic processes (frequent destruction of the septum), and tuberculosis of the nose. The term ozæna is now restricted to a well-defined characteristic disease,—chronic atrophic rhinitis. It essentially consists in the transformation of the cylindrical epithelium of the nose (especially that of the inferior turbinated bone) into numerous layers of corneous and decaying pavement epithelium with atrophy of the entire mucosa. In the course of time, the subjacent cartilaginous and bony parts also undergo atrophy.

The **etiology** of ozæna is still obscure. Possibly it is engendered by specific bacteria. The frequently present bacillus mucosa ozæna is, however, a saprophyte. Usually the disease develops only after the 10th–12th year, and is therefore seldom seen in children. By way of exception it has also been diagnosticated as early as the 3rd–4th year (Bouley). Frequently, parents, sisters, and brothers, suffer from the same disease which attacks with predilection persons having broad faces, broad noses.

Girls are more frequently afflicted than boys. The disease principally attacks anæmic and scrofulous individuals. Hereditary syphilis

is unjustly held responsible. Ozæna most frequently develops independently, at times also seeming to originate from a simple hypertrophic rhinitis.

The **symptoms** of ozæna in advanced cases are well marked. Usually, however, the patients are brought to the physician only when they annoy all their associates by the offensive odor. On examination the interior of the nose is found remarkably wide, so that without further trouble a large portion of the posterior pharyngeal wall may be surveyed. The fetid breath arises from a large quantity of secretion which often covers the entire nasal cavity with tough, sticky crusts. If these crusts are removed there is found underneath a liquid purulent secretion and a thin, smooth mucosa. In advanced stages, the highly shrivelled condition of the inferior turbinated bone is very significant. The patients bear their affliction with great equanimity; and as they frequently have lost their sense of smell they are unaware of the stench which emanates from the stagnated secretion. They are more apt to complain of the inability to remove the crusts from the nose, and of the troublesome pharyngitis sicca which constantly accompanies it. Ozæna cannot be cured, but the disease may be temporarily improved by rational treatment. At all events, the offensive odor may be made to disappear.

Diagnosis.—The diagnosis is made by direct inspection, but more accurately by the use of the nasal speculum and reflected light; ultimately, by aid of the probe. In young children it suffices to allow the reflected light to fall into the nasal cavity without the aid of a speculum by everting the tip of the nose. Marked tumefaction of the anterior portion obstructing the view usually subsides after pencilling with cocaine, whereby it is shown that one is not dealing with a true hyperplasia but with a simple congestion.

"Snuffles" during the early months of life demands a close search for other manifestations of syphilis. Foreign bodies cause a one-sided purulent discharge. Next to rhinitis, the most frequent cause of impeded nasal breathing is found in the presence of adenoids in the nasopharynx. Where the latter predominate, the secretion is generally more scanty, and in cases of extensive obstruction, in addition to defective speech, impairment of hearing is frequently present.

Chronic atrophic rhinitis is recognized by its extremely chronic course, its fetid discharge showing a strong tendency to dry and to form tough crusts, and by the spacious nasal cavity with atrophy of the mucous membrane and of the framework. Ulcerations, foreign bodies, abscesses of the accessory cavities (very frequent), are to be excluded. Mucous polypi are exceedingly rare, and are found only in older children. They are easily recognized by their roundish pediculated shape, their mobility, and by their semi-transparent, whitish, or pale-red, appearance.

The **prognosis** is fairly good. The simple and the hypertrophic rhinitis are almost always amenable to treatment, although transitions to the atrophic form (ozæna) may also occur. A rhinitis existing for years is detrimental to hearing and respiratory organs, and injures the development of the thorax and the facial skull.

The **prophylaxis** consists in building up the constitution, the combating of scrofulosis, and a general hygiene of the respiratory organs (described more explicitly under bronchitis).

Treatment.—Adenoid growths coëxisting with rhinitis are to be removed by operative measures. Nasal breathing being again rendered possible, the rhinitis is often cured in a short time without further treatment. Of great importance is the systematic removal of large accumulations of secretion. This purpose is served in the first place by a judicious method of blowing the nose.

The usual custom of pressing shut both alæ nasi, simultaneously, causes an injurious increase of pressure and stasis in the nose. If an attempt is made to have a child blow its nose in this manner, an uncommon dexterity is required on both sides in order to have the expulsion of secretion and the release of the alæ nasi occur simultaneously. The best method of blowing the nose is the one-sided one (like that of the working class, but with the use of the handkerchief). It is the only method which may be permitted in "the blowing of the nose" in young infants by a third person. The whole current of air is concentrated in the one half of the nose, and in this way expells the secretion much better. All injurious results from increased pressure are avoided, as well as the danger of forcing the secretion into the tubes.

A **cure** of rhinitis is often better and easier obtainable by attention to the general health, than by the use of local measures. Attention to cleanliness, suitable indoor climate, plenty of out-door life, suitable clothing, judicious hardening, and the avoidance of coddling, on the one hand, together with the provision of sufficient clothing, on the other, are to be taken into consideration.

In the many cases of chronic catarrh resulting from scrofulosis, the use of iron, iodide of iron and malt preparations, and codliver oil during the winter, will give good results. Strikingly beneficial are systematic hydrotherapy with the use of sea-salt or brine, residence at the seashore, or in forest regions and in the mountains.

As regards *local treatment*, the use of douches and injections had better be avoided, since they are usually met by considerable resistance on the part of children, and may lead to grave aural suppurations. For the last reason, also, the use of the Politzer air-douche for the purpose of removing secretions is inadvisable. Frequently, in chronic rhinitis, especially where the secretion is plentiful, good effects are obtained by evaporation of an ethereal oil in the vicinity of the patient.

For this purpose, 1–2 teaspoonfuls of the oil of turpentine are poured on a piece of pasteboard and placed near the head of the bed every night.

In older children, from five to seven years of age, irrigations of the nose with a lukewarm normal salt solution are advantageous, or when the secretion is tough, with sodium chloride, sodium bicarbonate, and sodium biborate, of each a knife's point-full to one glassful of water. The flushing is done by pouring the solution into the nose with a pointed teaspoon, the head resting slightly backwards. The solution runs through the lower nasal passage into the pharynx and is expectorated with the mucus which is carried with it. For the purpose of cleansing the nose, one can also use long tightly twisted pieces of cotton which are pushed horizontally backwards to the posterior nares.

Instillations into the nose of the oil of almonds with 1 per cent. of menthol, also a 3 per cent. aqueous solution of protargol, have often proved useful to me. 1–3 drops are instilled in each nostril several times. Insufflation of powders is popular, through insoluble powders must be avoided. To be considered are borax or boric acid with perhaps the addition of 10 per cent. sodium sozoiodol. For older children, the daily introduction of tampons spread with boro-vaseline are recommended for cleansing and healing purposes. In stubborn cases of rhinitis in older children the direct application of boro-vaseline often acts very beneficially (Boulay). The child introduces with the index finger into the vestibule of the nose a quantity of boro-vaseline (10–20 per cent.) about the size of a hazel-nut; the other nostril is then held shut and the boro-vaseline is snuffed up with the head bent backwards. It soon melts and lines the nasal cavity, producing a free discharge and softening the dried secretion. The other nostril is also attended to, at once. After 5–10 minutes the child should blow its nose, expelling the ointment and nasal secretions thoroughly. Good effects are often obtained in severe and obstinate cases of hyperplasia of the cushion of the mucous membrane by a 2–5 per cent. solution of nitrate of silver applied daily or every other day by the physician himself, the parts having been previously cleansed. Stoerk recommends the introduction of cotton tampons moistened with a 1–2 per cent. solution of nitrate of silver once or twice daily for several minutes. Highly hypertrophic portions of the mucous membrane are suitable for galvano-cautery treatment by the specialist. The often present eczema of the nasal orifices and vestibule of the nose is made to disappear by the application of 1–3 per cent. ointment of white precipitate ointment with the addition of lanolin, the crusts having been removed.

Chronic atrophic rhinitis demands treatment of the existing anæmia and scrofulosis. Locally, the frequent removal of the dried crusts is necessary. For this purpose, nasal douches carefully used and under

the control of the physician, are permissible (normal salt solution, sodium bicarbonate, 1 per cent. or thymol 1 : 10,000). The physician may also frequently remove the crusts with forceps or probe. In ozæna, especially, the treatment with boro-vaseline as described above, is said to be useful.

In obstinate cases, the crusts are loosened by the application of Gottstein's temporary tampon. Firmly rolled cotton tampons, dry or saturated with a 3 per cent. solution of peroxide of hydrogen, are introduced for $\frac{1}{4}$ to $\frac{1}{2}$ hour. They stimulate the secretion and carry the crusts with them. Insufflation of iodol on the cleansed mucous membrane is advocated by some.

EPISTAXIS

Etiology.—Nose bleeding of independent origin is rather rare in young infants, but in older children on the other hand it is frequently habitual, and may be classed with the numerous school maladies, being favored by a stooping position, tight collars, etc. Slight traumatisms and picking the nose are the most frequent contributing causes in older children. Local diseases such as rhinitis, foreign bodies, ulcerations, and adenoids, often cause nasal hæmorrhage. Severe infectious diseases, typhoid fever, sepsis, scarlet fever, and diphtheria (in the latter disease without the nose being always affected) may be accompanied by hæmorrhage. In severe cases of whooping-cough, the paroxysms are often accompanied by violent hæmorrhages from the nose. Of remote diseases, cardiac lesions, severe anæmia, general hæmophilic diathesis, are occasionally brought into play. In older girls, nose bleeding may occur vicariously in the place of the menses. During early infancy, nasal hæmorrhage is rare; in the newborn it usually points to syphilis or to a general sepsis.

Symptoms.—The bleeding usually takes place from one nostril only, and usually subsides spontaneously. The loss of blood is usually less than the attendants believe. Profuse, debilitating hæmorrhages occur usually only in the course of severe infections and in hæmorrhagic diathesis.

The **diagnosis** demands a knowledge of the cause, and, if possible, the site of the bleeding. For this purpose examination by means of the speculum is frequently required. In the vast majority of cases of habitual bleeding in older children, as in adults, the seat of bleeding is in the cartilaginous septum in front and below, therefore, at a point where it may be readily surveyed. The mucous membrane at this point is very thin, well supplied with dilated veins and capillaries which easily rupture. Frequently, a chronic catarrh in this situation (*rhinitis anterior sicca*) leads to brownish red, later more grayish discoloration of this part, with firmly adherent crustaceous secretion. The dry, itching,

sensation causes the children to pick the nose whereby the thin dry subjacent epithelium is often torn away and the blood vessels ruptured. After the cessation of the bleeding a small crust of blood is frequently seen at this place. Rarely does the bleeding take place from the floor of the nasal cavity or from the mucous membrane of the inferior turbinated bone. It is then often impossible to find the bleeding point. If the nose bleeding occurs during sleep or in the recumbent posture, the blood often flows backwards through the nares into the pharynx. Expelled by vomiting or coughing it sometimes is a source of alarm to the parents as "blood vomiting" or "blood coughing."

Prognosis.—In severe infectious diseases and in hæmophilia, hæmorrhage may persist until a stage of exhaustion dangerous to life is reached.

Prophylactic measures consist ultimately in overcoming the anæmia, and in forbidding in school children stooped positions and the wearing of tight neck-ware. An ointment of lanolin with boric acid (byrolin), or with 1–3 per cent. of yellow oxide of mercury or white precipitate ointment, acts very favorably where there is a tendency to bleeding from the anterior portion of the septum.

Treatment in mild cases, that do not frequently recur, is unnecessary. The usual popular remedies (cold compresses to the back of the neck, elevation of the hands) are of no avail, but have the advantage of causing parents and children to await with more patience the cessation of the bleeding which usually soon occurs. The snuffing up of cold water, or the introduction into the nose of small pieces of ice is often useful. Blowing the nose should be avoided. Hæmorrhage, occurring as the result of severe overheating or cephalic congestion, often causes great relief. The child should sit before a basin with the head bent forward so that the blood drops into the vessel, and should be instructed to take deep nspirations by which the bleeding is usually caused to cease promptly.

In most cases the bleeding is stopped as soon as the ala of the affected side of the nose is pressed against the septum with the finger, whereby the bleeding point is compressed.

The bleeding is most effectually stopped by tamponing the affected side of the nose. The most simple remedy and one which can easily be taught the parents is the introduction of a cotton plug. A piece of absorbent cotton about 3–6 cm. in length is twisted on a cotton carrier or a flattened wire, exactly of the thickness that can just be forced into the nose. It is rotated horizontally and pushed firmly backwards, the tip of the nose being raised. This simple remedy nearly always suffices.

If the purpose is not attained by this alone, a tampon may be saturated with a 20 per cent. solution of antipyrin, or 5 per cent. potassium permanganate; a dry tampon may also be dusted with antipyrin or powdered ferri-pyrin. Chloride of iron is a good styptic, but on account of its injurious and corrosive action should not be employed. The

application of Penghawar-Jambi cotton is recommended. If, after sponging with cotton, the bleeding point is seen (which is easily done when it occurs at the point of predilection anteriorly on the septum), the physician can stop the bleeding by direct cauterization of the point with the nitrate of silver stick. Here, also, according to Siebenmann, good hæmostatic effects are obtained by pressing against the bleeding point a pledget of cotton dusted with powdered potassium permanganate. Recently, also, adrenal preparations are often used; adrenalin 1 :1000, introduced on small pledgets of cotton.

Complete tamponage is required only in extremely rare cases. Narrow strips of gauze are introduced (after cocainization if necessary) along the floor of the nasal cavity to the posterior nares until the nose is completely plugged. The torturing Bellocq's cannula which still enjoys its undeserved popularity is, at the present day, also forbidden by most specialists. In unusually obstinate and violent hæmorrhage, subcutaneous injections of 20–50 Gm. of a 10 per cent. solution of gelatin (sterile on account of danger from tetanus) should be tried.

FOREIGN BODIES

Occurrence.—Children from three to five years of age like to shove small objects into the nose (pebbles, glass beads, peas, beans, cherry seeds, paper balls, etc.). More rarely, foreign bodies get into the nose through the posterior nares by choking or coughing (pieces of bone, fruit seeds, etc.). Occasionally, so-called rhinoliths develop on the spot, *i.e.*, concretions of carbonate and phosphate of lime which may adhere to a small foreign body or perhaps merely to a blood clot. Only very seldom in our region are found the larvæ of flies (Peiper) which may produce serious cerebral disturbances.

Symptoms.—At first, there exists only a simple obstruction of the affected half of the nose, which often does not inconvenience the child, but which may give a nasal intonation to the voice. Since children often seek to conceal the fact from fear of punishment or at first may not even notice it, the foreign body may remain undiscovered for days, and smooth, round, impervious objects, even for years. Angular objects, especially those which are pervious and capable of swelling (peas, beans, though usually only after a short period), cause a local inflammation which may lead to an offensive, bloody discharge, ulceration, and formation of granulations. It may also lead to purulent otitis, general infection, and cerebral manifestations.

The **diagnosis** is easy, if the child is brought by the alarmed parents to the physician, immediately after the occurrence. The foreign body usually rests far forwards in the nose, usually in the middle nasal passage or between the inferior turbinated and the septum. From awkward attempts at extraction it has often slipped further backwards.

Very often the physician sees the patient only after 1–2 weeks, or even very much later, when more inflammatory reaction has set in. A *one-sided, purulent, offensive discharge in a child almost always points to a foreign body,* as abscess of the accessory cavities is of rare occurrence. Careful cleansing, cocainization, and examination with the probe, is then often necessary for diagnosis.

The **treatment** of the foreign body is simple. Very frequently, the physician can force it out by manual pressure on the wing of the nose. In older children, blowing the nose vigorously with closure of the free side, is at times sufficient to cause the expulsion of the object; but in younger children it is often necessary to use a Politzer air-douche to the free nasal side. If the foreign body is wedged in, it is worked out by means of a heavy probe which is bent at a right angle at its extremity for about 1 cm. It is passed from above to the rear of the foreign body and drawn out with a vigorous jerk. Ultimately, for the reduction of the swelling, cocainization will be found very useful. Occasionally, a wire snare, or a tenaculum forceps, will grasp the object better than the bent probe. If the object is in the rear portion of the nose, whence it is often impossible to bring it forward, it is then pushed backwards while the velum palati is raised with one index finger, so that the foreign body cannot possibly drop into the larynx. In obstreperous children bromethyl narcosis is often required.

TRACHEAL AND BRONCHIAL CATARRH (BRONCHITIS)

Occurrence and Etiology.—Catarrhal affections of the wind-pipe and of the bronchial tree are found with extraordinary frequency during the entire period of childhood, *especially at the ages of six months to three years.* Uncleanliness, poor sunless dwellings, lack of care (constant dorsal position in infants), promote its development to a very great extent. Abrupt changes in the weather, with heavy rains and raw winds during the cooler season of the year, produce an increased number of cases at this time. The prolonged stay in closed and heated rooms during the winter,—lowering the vitality more and more,—reinforced by the inclemency of the spring-weather, cause the majority of cases to develop during this season of the year. The infectious diseases rapidly increase in number during October, reach their maximum during November and December, and decrease very rapidly again during April (Homburger). Colds exercise considerable influence. The normal mucous membrane of the trachea and the bronchi destroys the majority of the invading germs, and harbors only a few attenuated pathogenic bacteria (Dürck, Müller). The mucous membrane, weakened from whatever cause, is no longer capable of preventing the increase of the on-rushing bacteria; while in severe infections (measles, influenza), the safeguards of the normal mucous membrane also fail.

The acute catarrhs depend mostly upon infection with very many different kinds of bacteria, particularly with pneumo- and streptococci (A. Fränkel). The contagion so frequently observed is thereby readily explained. Like Müller, I also have frequently observed in private practice, that the same infection in different members of the family evidently causes coryza, angina, bronchitis, or intestinal catarrh (especially in infants), according as the case may be. The specific catarrhs in the course of many infectious diseases (measles, whooping-cough, influenza), frequently undergo secondary infection. The contagious domestic grippe, occurring regularly and often epidemically during the cold season of the year, chiefly runs its course with manifestations of bronchitis (epidemic bronchitis). I agree with Filatow and Stooss, that the grippe, which probably depends upon pneumococci (Stooss, Luzzato) and which preferably attacks children (infantile grippe) is not identical with pandemic influenza with which it is mostly classed. This also explains why the influenza bacillus frequently fails to be found in "influenza."

The *chronic bronchial catarrhs* frequently arise in connection with chronic affections of the nasopharynx, very often in mouth-breathers (adenoids), or accompany diseases of the lungs. In these instances, infection plays a rather unimportant and secondary rôle. Of much more importance are the anatomical changes in the mucous membrane and the constitutional anomalies, anæmia, rachitis and scrofulosis. While the majority of German authors are unfavorably inclined thereto, much importance is attached by the French to the gouty diathesis, the nemo-arthritismus, and especially also to a closer connection between chronic skin affections (eczema, prurigo, scrophulus) and bronchitis, particularly the asthmatic form, a fact which has been given prominence, with others, both by Heubner and myself.

According to my observations, I regard dentition as blameless in the etiology of the disease in question, and moreover in the whole pathology. The still great popularity of dentition as an etiological factor with physicians and laymen may be accounted for by the great convenience and comfort which it affords to one or both parties.

Disturbance of the pulmonary circulation, mostly due to mitral lesions, leads to statis bronchitis. The frequent occurrence of bronchial and pulmonary affections with intestinal diseases is attributed without sufficient proof to coli infection by Lesage and Sevéstre.

Pathological Anatomy.—In acute catarrhal inflammation of the trachea and the bronchi the mucous membrane is swollen, loosened and reddened from the dilatation of the small blood vessels. The mucous secreting goblet-cells are markedly increased, and likewise the deeper lying coils of the mucous glands. The subepithelial layer and often also the submucosa are greatly infiltrated by round cells which partly wander

to the surface of the mucosa. The secretion of the coarser bronchi is more slimy, that of the finer bronchi more purulent. The bacterial content is very large.

In *chronic bronchitis* the mucous membrane is brown or grayish red, often thickened from proliferation of interstitial connective tissue. Fibrinous peribronchitis, dilatation of the bronchi, swelling of the bronchial glands, are frequent findings, and more rarely, emphysema.

Symptoms.—Catarrh of the trachea scarcely ever occurs isolated, usually being found in connection with catarrh of the larynx or of the bronchi. It is customary to distinguish bronchitis without fever, and bronchitis with fever, although they cannot be sharply defined, and many transitions exist. In the following short description, only simple bronchitis will be considered. Disease of the finer bronchi (capillary bronchitis) is described in the following chapter.

Acute bronchitis generally develops rapidly after an analogous affection of the air-passage above, or simultaneously with it, and manifests itself by cough and fever of a varying degree. In some individuals, especially during the first years, a descending bronchitis regularly sets in after each attack of coryza.

The cough in acute bronchitis is at first frequent, dry, and irritating, it is also present at night, and is at times paroxysmal (frequently so in grippe); later it is loose and less frequent, and no longer disturbs the night's rest. It is often accompanied by pain in the chest anteriorly.

Expectoration is usually absent in children under seven to ten years in all bronchial and pulmonary affections. It is seen only in younger children after vomiting or in experienced coughers; for instance, if preceded by a protracted siege of whooping-cough. Infants swallow the expectoration as soon as it is thrown against the roof of the pharynx by the cough.

On careful examination the respective movements of swallowing may be seen after severe fits of coughing. Prolonged coughing in the morning on awakening is usually the sign of free secretion. If the sputum of an infant be desired, it can be easily obtained with a sterile cotton swab, passed through the opened mouth during an attack of coughing. It is also readily obtained by *siphoning* the *empty stomach*. The expectoration, as in the adult, is at first glassy and tough, later mostly mucopurulent. Seldom and only in older children "dry catarrh" is met with. Where the expectoration is offensive, it is spoken of as fœtid bronchitis; frequently complications of bronchiectasis, foreign bodies, or gangrene of the lungs, are present. At times the temperature reaches as high as 40° C. (104° F.), but usually declines after a few days. The elevation and duration of the temperature do not depend so much on the bronchitis alone, as, often in a great measure, on the accompanying general infection (for instance influenza, grippe). Long-continued or repeated

elevations of temperature mostly depend on the bronchiolitis that sets in, or on complications (otitis, etc.).

The *respiration* often remains unchanged in a febrile bronchitis, and is increased and labored only when there is much accumulation of mucous or when emphysema is present. When fever is present, it is often difficult to decide to what extent the increased respiration is due to the elevation of temperature or to the bronchitis. Marked dyspnœa or cyanosis, increased inspiratory recessions, usually point to the presence of a capillary bronchitis. Only in nurslings are inspiratory recessions seen, even when the catarrh is limited to the coarser bronchi merely, and they stand out more prominently during crying or coughing.

The circulatory organs usually present nothing remarkable except a moderate acceleration of the pulse which is correspondingly increased by the height of the fever. In the course of whooping-cough demonstrable dilatation of the right heart frequently develops.

Physical examination often gives cognizance of whizzing ronchi on palpation of the thorax, which may also be heard at a distance. Pain on lifting up under the arms frequently proceeds from a florid rachitis, and only exceptionally from a dry pleurisy. At first auscultation is negative as long as the catarrh is dry, and limited to the trachea and the very large bronchi. Soon scattered purring and whizzing ronchi appear, later moist, coarse, and medium râles. The vesicular breathing is often accentuated, expiration prolonged. Percussion shows normal conditions except in bronchial asthma and in emphysema.

During the febrile period, the general condition is often very much affected by headache, malaise, thirst, disturbance of the appetite, and sleep. In younger children, diarrhœa often sets in.

Simple bronchitis terminates in recovery in from two to six weeks on an average. However, during the early years, the occurrence of bronchiolitis, bronchopneumonia, and also other complications (otitis, gastro-intestinal disturbances), are frequent and dangerous.

Chronic bronchial catarrh develops gradually or follows protracted acute or relapsing catarrhs. A peculiar form of chronic tracheal and bronchial catarrh often occurs in nurslings and is often seen even during the early months (Henoch). Associated with a frequent, initiating cough, an inspiratory and expiratory stridor is heard at a distance (rattling in the chest), which temporarily disappears after an attack of coughing. Auscultation reveals coarse or sibilant râles, especially between the shoulder blades. Fever is absent. The general condition is undisturbed, and infants often look pale and shrivelled. I, also, can vouch for the frequency of this very obstinate form of catarrh which is also often found in pasty looking infants.

Membranous or plastic (fibrinous) bronchitis is a disease the chief characteristic of which is tubular, white or yellowish white exuda-

tion on the mucous membrane of the trachea or of the bronchial tree. Leaving out of consideration the frequent diphtherial bronchitis, bronchial croup (see diphtheria) in its limited sense, one not infrequently finds in croupous pneumonia, a membranous coagulum formation in the finer bronchi emanating from the alveoli. Besides these two diseases which we do not consider here, membranous bronchitis rarely appears, and is in children especially seldom met with, probably only from about the fourth year on.

An acute and chronic form are distinguished. The acute form seems to be dependent on bacteria; pneumococci (Jaccoud, Marfan), pneumobacillus of Friedlaender (Magniaux), streptococci (Claisse), are held responsible. The patients suffer from fever, irritating cough, dyspnœa, and cyanosis. After expectoration of conglomerated dendritic coagulum, great relief often occurs which frequently, however, is only temporary. Relapses may occur after days and weeks, and terminate either in recovery or in death from asphyxia and exhaustion.

The chronic, relapsing form produces attacks similar to the acute, which recur from time to time for years and years. The expectorated matter does not consist of fibrin but mostly of mucus, and is so-to-speak sterile and often contains eosinophile cells, Charcot-Leyden crystals, and Curschmanns spirals. Accordingly, the disease may be regarded as a possible neurosis of secretion in favor of which are the occurrence, not uncommonly, of asthmatic conditions during the intervals of freedom.

The **diagnosis** is to be made by the tubular, dendritic, branched, casts. Marked inspiratory recessions, impeded expiration, cyanosis in the absence of bronchiolitis, and normal percussion note, with reduced thoracic movements, must lead one to think of this rare affection.

The **prognosis** in the acute cases is mostly fatal; but in the chronic form life is less endangered, though lasting recovery is seldom to be expected.

The **treatment** requires trial of sweat-cures, inhalations of alkaline waters, and especially potassium iodide. Lastly, where the membranes are loosened an emetic is to be considered.

The *diagnosis of bronchitis* depends on the presence of non-metallic coarse and medium or sibilant râles, with normal percussion note, absence of bronchial breathing, and bronchophony. Involvement of the trachea often produces, on coughing, a burning pain behind the sternum, which cannot be physically diagnosed (without a reflection). Coarse râles or wheezing equally perceptible over both lungs may be located in the trachea or in the neighborhood of the bifurcation. Frequently a cough with negative auscultatory findings is diagnosed as tracheal catarrh where merely a pharyngo-rhinitis is in question. Coarse râles originating during crying, and accumulation of mucus in the nasopharyngeal space, may readily pose as bronchitic signs to the inexperienced.

In cases of continuous high temperature with absence of bronchitic signs one should think of central croupous pneumonia. Confusion with whooping-cough and tuberculosis of the bronchial glands is often to be considered. One must think of whooping-cough especially whenever there is present an obstinate dry cough without bronchitic signs, gradually increasing, assuming a spasmodic character, and also appearing at intervals during the night.

Presenting the same picture or associated with chronic bronchial catarrh, tuberculosis of the lungs at times develops, whereby, owing to the absence of sputum, the distinction is rendered more difficult. It is also of importance to diagnose the cause of the bronchitis; for instance, whether it has arisen independently (colds, local infection, mouth-breathing) or occurs as the result of rachitis, pertussis, grippe, etc. (in this instance often the only manifestation of the disease).

By *periodical night-cough* is understood attacks of coughing in older children, awakening them from their sleep, and in some cases occurring regularly and lasting for several hours. The finding in the lungs is entirely negative. The disease is mostly of a nervous character and is cured by quinine or bromide. Frequently, however, an unrecognized nasopharyngeal catarrh lies at the basis.

The **prognosis** in older children is good. In infants with florid rachitis it is doubtful, especially when bronchitis occurs as an accompaniment of some febrile disease or pertussis.

Prophylaxis exercises an extraordinary influence. Nasopharyngeal affections are to be carefully treated, and rachitis and scrofulosis energetically combated. Intercourse with those affected with colds and sore-throats is to be strictly avoided. Should the mother or nurse be thus afflicted, she must not kiss the child and must turn her head away while coughing. Beyond this, the general prophylaxis embraces the whole hygiene of the infant; but only the most important points pertaining to the diseases of the respiratory system will be especially mentioned here.

Of primary importance is the unlimited supply of *fresh air* not only by plenty of outdoor exercise but in the house as well, the poor ventilation of which produces the majority of the diseases of the respiratory system. The best and sunniest rooms where no heavy curtains shut off the light should be used for children, and provision made for frequent changes of air. During the period of heating, the temperature of the room for nurslings should be about 15° R. (66° F.); while for older children the living room should be 14°–12° R. (64°–59° F.), and the sleeping apartment 12°–8° R. (59°–50° F.). Children should be accustomed to sleep with the windows open during the summer months, and for older children with sufficient covering for the upper portion of the body the windows may be left partially open during the winter. The

best method of heating is with tile stoves, as they also ventilate well when the lid is allowed to remain open after the room is heated. Hot-water heating is good, in so far as provision is made for the necessary moisture. During the winter, moreover, the air of the room usually becomes too dry (a chief cause of many catarrhs), and must be artificially moistened by placing a basin of water on the stove, or more effectually by hanging up dampened cloths (50–70 per cent. of moisture is desirable). The air must have free access to the bed; curtained beds are detrimental. Large carpets are dust catchers and are not to be tolerated in children's rooms. The clothing must be adapted to the season of the year and to the weather and should not be too warm nor too light. During winter, woolen stockings, and for many children woolen waists, are of advantage. Drawers which are too thin are frequent deficiencies of the customary dress during the first years. Warm petticoats in children who frequently sit on the floor, are no substitute for them. If, after going out during the winter, covered portions of the body feel cold to the touch, the clothing has not been warm enough. The custom of having the limbs bare is responsible for frequent colds. For the care of the skin, in addition to sponging and baths, dry rubbing with the hand or cloth is very useful. In children over two years of age after a warm bath, and before drying, the skin should be quickly sponged with a wet and cold sponge. From three years on, for the purpose of hardening, frictions of the whole body with a wet cloth (temperature of the room), or later douches from spout of a sprinkling-can, are often useful. The procedure should take place in the morning immediately on arising, should last only one quarter of a minute, and should be followed by energetic friction with a dry cloth. Hydriatic hardening is not suitable for many of the younger and feeble children, especially if reaction of the skin remains absent. Hecker properly emphasizes the disadvantages of routine "hardening" which often brings about the opposite result. There is need of careful watching, and prudence and practice are required, a difficult art, naturally. Clothing which is not too warm, moderately heated rooms, and the custom of going out even when the weather is unfavorable, often increase the power of resistance much more than the so-called hardening.

Treatment.—In very early cases, the disease may often be cut short by free diaphoresis (hot teas, and if necessary, the simultaneous administration of aspirin).

In acute bronchitis, it is proper to keep the patient in a room with an equably warm atmosphere. Rest in bed is necessary when fever is present, or even when there is only an evening elevation of temperature.

Whenever the atmosphere of the room is excessively dry, as is especially the case during the time when the rooms are heated, provision must be made for a necessary amount of moisture by evaporation

of water, particularly in cases of infants. For this purpose, in severe forms and especially in those frequently associated with catarrhal laryngitis, the croup bronchitis-kettle (see Fig. 69, page 348) is useful. The trunk of the patient is covered with a woolen or, better still, flannel jacket. It also suffices to cut two holes for the arms in a suitable piece of flannel. For the relief of the cough and stimulation of the secretion the drinking of alkaline muriatic waters, marshmallow tea, with the addition of extract of malt, is of service. Older children may be given inhalations of common salt (12 to 15 Gm. (3–3¾ dr.) to 1 litre (1 quart) of water). Mild cases recover without further treatment.

Hydrotherapy is most efficacious in high temperature, severe bronchitis, or long continuance. In febrile bronchitis compresses saturated with water at the temperature of the room, are applied to the chest and changed every one or two hours; in older children, when the temperature is high, every half hour, or, for a short time even every quarter of an hour. Rubber tissue is not used for this purpose. Patients with high temperature and severe constitutional disturbances may be given tepid baths, 27° R. (93° F.) for infants, 25° R. (89° F.) for older children, from 4–6 minutes morning and evening. In cases of great restlessness and fever during the night, a cold compress applied from the arm-pits to the middle of the leg for fifteen minutes is of value. As the temperature declines the compresses are changed only every two or three hours, gradually going over to lukewarm. (Compare with temperature of baths and packs and also with what is stated on page 350.)

In acute afebrile bronchial catarrh warm compresses are used from the beginning and are changed every two or three hours during the entire day, or only one compress is applied for about three hours each morning and evening. Friction of the skin and of the body with dry or cold wet cloths when the compresses are changed is of utility. In cases of feeble constitution, cool skin or cold rooms (among the poor), it is well to introduce rubber tissue between the wet chest compress and the covering.

After removal of the compress the body must be covered with a dry woolen jacket. Frequent changes of position and taking up out of bed is necessary in young infants for the purpose of increased aëration of the lungs.

Method of Applying the Compress.—The application of the chest compress must be first demonstrated and supervised by the physician. One or two towels or a diaper are folded four or eight times in such a manner that the breadth reaches from the axilla to the umbilicus, and the length somewhat exceeds the chest circumference. It is then dipped either in cold or hot water, firmly wrung out, and quickly spread smoothly on a previously prepared flannel blanket (for which purpose the baby shawls are well adapted), and the whole quickly wrapped around the

chest of the patient from behind, as high as possible in the axilla, permitting the arms to remain free. With some practice this is readily done without removal of the infant's shirt. At first the free ends of the wet cloth, and afterwards those of the dry, are crossed on the front of the chest and the blanket firmly fastened with safety-pins. The outside blanket should extend for one or two finger-breadths beyond that of the underlying wet cloth so that nothing can be seen of the wet cloth. The compress must be made so that the moisture does not penetrate the outside covering. When the compresses are to be changed continuously two cover blankets are required so that each one may be thoroughly dried during the intervals. If warm compresses are desired, the cloths are immersed in hot water, spread out, and immediately applied as soon as they have cooled off to the desired temperature. For the purpose of an impermeable covering, Billroth's batiste or something similar, between the outside cover and the wet cloth, is advisable.

Febrifuges are almost always superfluous and frequently harmful. As soon as the catarrh begins to resolve, or even in the beginning, the evaporation of ethereal oils is advantageous, oleum pini pumilion., ol. eucalypti (10–30 drops frequently dropped on a cloth). The common oil of turpentine is also good and much cheaper. One or two tablespoonfuls of this are poured on a piece of cardboard or blotting paper, once or twice daily and placed in the vicinity of the patient. Inunctions of the chest once or twice daily, with a mixture of equal parts turpentine and olive oil are frequently useful.

One must be cautious regarding the use of the favorite expectorants. In a beginning dry catarrh, ipecacuanha acts well (0.1–0.4 per cent.) One teaspoonful of the infusion can be given every two hours, and in constipation powdered ipecac, 0.01 ($\frac{1}{6}$ gr.) with calomel 0.01–0.03 Gm. ($\frac{1}{6}$–$\frac{1}{2}$ gr.) five doses to be given, or extract ipecac fluid 0.1–0.3 : 30 ($\frac{1}{6}$–$\frac{1}{2}$ gr. to 1 oz.) 20 drops 3 times daily. In favor also are ammon. chlorat. (1–2 : 100 with 3.0–5.0 c.c. syrup) and apomorphine muriat. 0.01–0.05 : 100 ($\frac{1}{6}$–$\frac{3}{4}$ gr. to 3 oz.) the latter especially in involvement of the larynx. In asthmatic catarrh (see bronchial asthma), sodium iodide 1.0–3.0 : 100 (15–45 gr. to 3 oz.) with syrup, not to be repeated. If extensive mucous râles are heard, liq. ammon. anisat. (P. G.) 2–10 drops 3 times daily, or elix. e. succo liquirit 10–40 drops in sugar water 3 times daily are given as expectorants. They may also be added to other mixtures. Where there is an accumulation of secretion a more decided expectorant action may be obtained from senega in 3–5 per cent. decoction. One must seek to limit the continued formation of mucus by guaiacol carbonat. 0.5–0.25 ($\frac{3}{4}$–4 gr.) 3 times daily or creosotal, terpin hydrate, etc.

Only *when the secretion is scanty may the irritative cough* be allayed. When extensive mucous râles are present with an excessive amount of mucus in the bronchial tubes the cough must even be energetically stim-

ulated by liq. ammon. senega, compresses, etc. Free expectoration is under these circumstances the best method of lessening the cough. Morphine is given only to older children, after the sixth year [2–5 milligrams ($\frac{1}{32}$–$\frac{1}{12}$ gr.) per day]. Usually, it may be dispensed with. Codeine phosphoricum acts pleasantly and may be given in doses of 0.01 Gm. ($\frac{1}{6}$ gr.) per day to a four-year-old child, and 0.02–0.04 Gm. ($\frac{1}{2}$–$\frac{3}{4}$ gr.) to one six to ten years old. During the early years these remedies had better be avoided. To allay the cough rather give aqua amygdal. amar. (3 times daily, as many drops as there are years) added to the medicine or ammon. bromat. 1.0–2.0 : 100 (15–30 gr. to 3 oz.). Bromoform (4 times daily, as many drops as years) also relieves the harassing cough or inunctions of antitussin, very frequently also ext. belladonn. 0.03–0.05 : 50 ($\frac{1}{2}$–$\frac{3}{4}$ gr. to $1\frac{1}{2}$ oz.) 5 drops 2–4 times daily. The narcotic drugs are usually added to expectorant mixtures.

During the febrile period the diet should be limited to fluids (milk, barley water, and for older children also gruels, eggs, and zwieback), with exclusion of all highly seasoned foods. Later, softened rolls, cocoa, rice, raised pastry, cooked fruits, finely cut roasted meat, may be given. Drinks should be freely allowed, but must only be given warm. In younger children, disturbances of digestion are frequently produced by energetic internal medication, the occurrence of which should lead to the suspension of all internal remedies as far as possible. After recovery from bronchitis, a sojourn in the country, or in the mountains, or at the sea-shore, is advisable; during the winter a stay at Lake Geneva, Riviera, etc.

In *chronic bronchitis* warm or hot chest compresses with rubber sheeting applied for three hours, once or twice daily, render very good service. In addition to a liberal diet, preparations increasing the blood formation and stimulating the appetite are indicated. Woolen underwear and stockings are indispensable in middle European climates. By cold sponging in the morning, and dry frictions, one seeks to attain hardening. Existing fundamental diseases must be removed (in rachitis, phosphorus and salt baths). Expectorants rarely give much relief, and are only used in acute exacerbations and excessive accumulations of mucus. Of much more value in excessive secretion are the tar preparations: guaiacol carbon. 0.1–0.25 Gm. ($1\frac{1}{2}$–4 gr.) three times daily, creosotal in emulsion 5.0–10.0 Gm. : 250.0 c.c. a dessertspoonful three times daily, aqua picis, a teaspoonful to a tablespoonful three times daily, terpin hydrate 0.1–0.5 Gm. ($1\frac{1}{2}$–7 gr.) three times daily, or ol. pini pumil. 2–8 drops three times daily in milk. The last also acts well in fœtid bronchitis. In addition, in older children, where the secretion is excessive, free evaporation of turpentine and inhalations of aqua picis, 1 : 10–1 : 2, are of value. In dry catarrhs, the prolonged use of inhalations of common salt or Ems salt (or with the artificial salt), are often

useful, as are the drinking of mineral or sulphur waters. Brine baths either at home or at the resorts are frequently attended by beneficial results. As a general rule, the most lasting effects are obtained by prolonged and repeated stays in forest regions, free from dust and protected from the wind or at the sea-shore. If possible, the patients should be sent during the summer to the mountains (for instance to the Black Forest, Flins, Engelberg); and more robust individuals even to the high Alps where even in winter excellent recoveries are obtainable (Arosa, St. Morrvitz), especially in anæmia and imperfect development of the chest. More delicate patients are to be sent during the winter to the Riviera, Isle of Wight, Algeria, or Madeira. A sojourn at the sea-shore, North and Baltic Seas, Wyk on Föhr, Abbazia, etc., is often beneficial, even in winter, and acts very favorably on the accompanying naso-pharyngeal catarrh.

CAPILLARY BRONCHITIS AND BRONCHOPNEUMONIA (CATARRHAL PNEUMONIA)

Bronchitis is apt to involve the finer and the finest bronchi, and is then designated as bronchiolitis or capillary bronchitis. With unusual frequency the disease extends from here to the lung tissues proper, and in this way leads to a bronchopneumonia (catarrhal pneumonia) which may also be described less accurately as "lobular pneumonia." The bronchopneumonia invades the lung tissue with numerous scattered nodules about the size of peas or nuts, which are principally found in posterior inferior portions, and which, by increase and spread to the intervening tissues containing air, may steadily cause larger portions to become consolidated. The course of the disease is acute, subacute, or chronic.

Since the treatment of capillary bronchitis and of bronchopneumonia coincide, and the clinical picture and the pathological anatomy have much similarity and present inseparable transitions, a joint description is advisable.

Pathology.—Capillary bronchitis presents an enormous dilatation of the corresponding blood vessels. The lumen of the bronchiolis is diminished by the greatly swollen mucosa and occluded by purulent and occasionally sanguinous exudate. The occlusion of the bronchioles leads to atelectasis of the alveolar areas, which formerly was regarded as the chief cause of the development of the bronchopneumonic areas. The pneumonic areas do not develop as the result of direct extension of the inflammation from the bronchioles to the alveoli, but by spread of the inflammation from the walls of the bronchioles and through the walls themselves to the surrounding tissue (Aufrecht), which is markedly infiltrated with lymphocytes.

An inflammatory œdema now extends from the septa of the alveoli to the neighboring alveoli, leading to colonization of bacteria in this re-

gion, to casting off of alveolar epithelium, and to distention of the alveoli with white blood corpuscles. A small quantity of fibrin is also found in the alveoli but never in the same amount as in croupous pneumonia. Frequently also red corpuscles are found in varying numbers. In the beginning, bronchopneumonia produces areas of peribronchitis varying from about the size of the head of a pin to that of a pea, which surround the pus-distended bronchioles between which lie areas that are still intact and that contain air (see Plate 47). The areas gradually become more numerous and increase to the size of a nut. Becoming confluent, they may involve an entire lobe. As a rule, the bronchopneumonic areas are dark blue in color and tough, later becoming grayish yellow in appearance with prominence of the infiltrated lobuli. On pressure, thick pus oozes from the bronchioles which are partly dilated. In addition to the pneumonic areas, as a result of the shutting off of the air, larger and smaller areas of atelectasis are to be found, especially in the dependent portions of the lungs. In the later stages, the connective tissue around the bronchi is very much increased (interstitial peribronchitis), leading, in some cases, to obliteration of the bronchi and the lung tissue. In addition, gangrene and abscesses of the lungs may occasionally result. The bronchial and tracheal lymphatic glands are enlarged (see also Plate 48).

Etiology.—In general, the etiology is identical with that of simple bronchitis. The disease occurs in connection with inflammatory conditions of the upper air-passages or simultaneously with them, very often in certain infectious diseases especially, frequently in measles, whooping-cough, grippe, influenza, diphtheria, and often also, as an accompaniment of severe intestinal disturbances. Some authors are of the opinion that certain forms of bronchopneumonia are contagious.

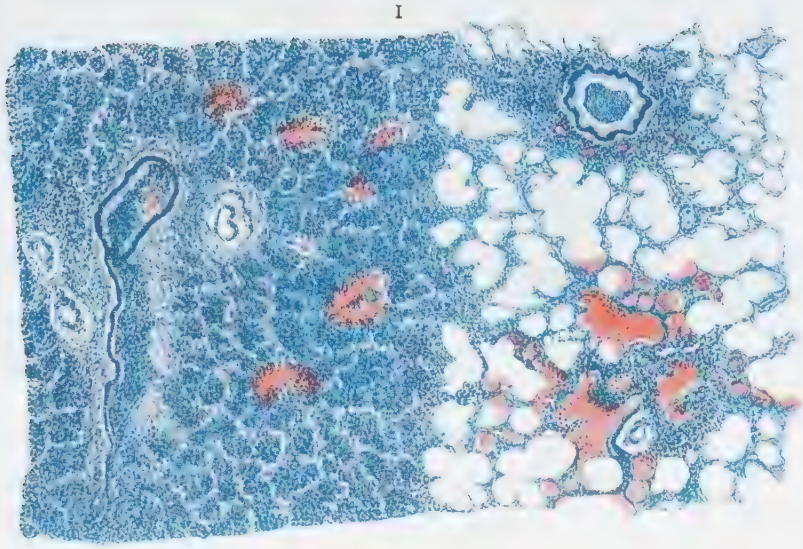
Capillary bronchitis and bronchopneumonia are preëminently diseases of early life (*infantile pneumonia*). They become less frequent after the fourth year. The largest number of cases are seen between the ages of six months and two years. The disease probably always is of an infectious character. Occasionally the bacteria of the primary diseases are found in the bronchioles or alveoli (diphtheria, influenza, typhoid), frequently in mixed infection. The most frequent findings are Fränkel's pneumococci, often in pure cultures, especially in primary cases, and streptococci (always in diphtheria), and often Friedländer's pneumo-bacillus, staphylococci, etc. (Netter).

From what has been mentioned, bronchopneumonia is not a disease *sui generis*, like croupous pneumonia, but is a secondary manifestation which, like the causative bronchiolitis, may occur in connection with different kinds of affections.

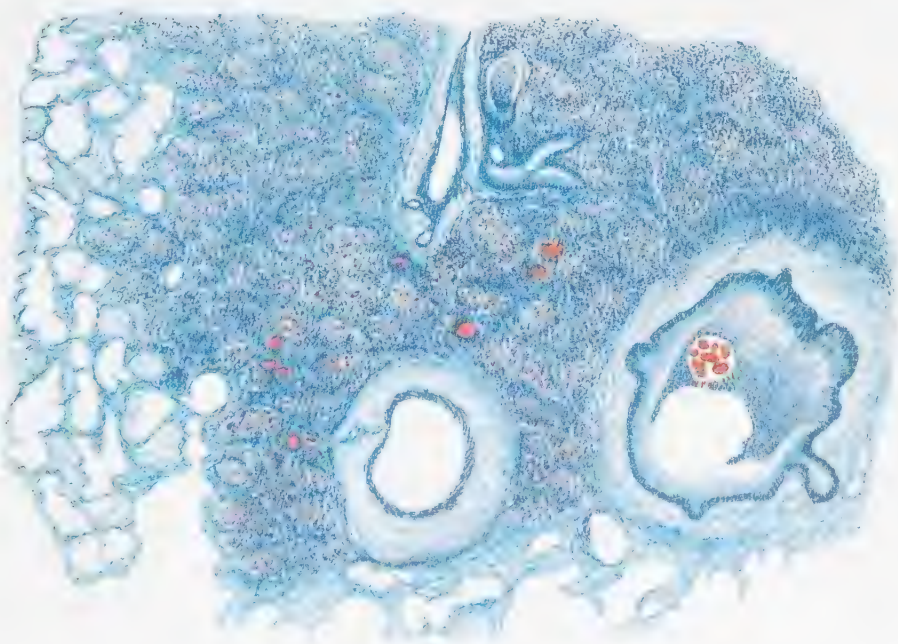
General Course of the Disease.—Bronchiolitis most frequently develops in connection with a catarrh of the median bronchi. Its onset

PLATE 47.

I



II



PNEUMONIA.

I. Typical pneumonia of infants. The hæmorrhagic exudate is stained red.
II. Aspiration pneumonia. Fat-globules from milk are stained red.

is announced by elevation of temperature, by severe coughing, and especially by dyspnœa. The respiration is increased and becomes labored, the number of respirations reaches 50–60 in younger and 60–80 in rachitic children. The nostrils and auxiliary muscles are brought into action during inspiration. Inspiratory recessions appear in the sternal notch and at the lower portion of the thorax, especially along the attachment of the diaphragm (peripneumonic fissure), an important sign showing that the access of air to the lungs through the diminished or obliterated bronchioles is rendered very difficult. The cry is short and suppressed, the pulse is much increased, 120–180, and small. The inadequate supply of oxygen soon leads to cyanosis (most plainly visible at the lips and finger-nails), and to serious disturbance of the appetite and general health. All smiles fade from the pale anxious face. The little patient restlessly throws himself from one side to the other. At a glance the experienced observer recognizes a serious disease of the organs of respiration. The physical examination reveals, besides medium râles which have probably already been present for many days, scattered fine subcrepitant râles most frequently in the lower inferior portions of the lungs. Percussion shows a normal condition or emphysema of the anterior portions of the lungs. The acute emphysema of the anterior portions of the lungs particularly of the upper lobes is a special peculiarity during early years (Gregor). The ronchi are not so uniform as the crepitant râles, are chiefly inspiratory, but are also often heard in the beginning of expiration. The vesicular murmur is often diminished almost to the point of disappearance. Bronchiolitis subsides after a period of several days or weeks (mostly after one or two weeks), with gradual abatement of fever and dyspnœa, the fine râles becoming less and less distinct. Very frequently, however, it leads to a fatal termination by the increasing occlusion of the bronchioles, sometimes without the presence of a bronchopneumonia, but mostly only after the development of the same. There is no sharp distinction, and often where a capillary bronchitis could be diagnosticated during life, on post-mortem examination numerous scattered bronchopneumonic areas are to be found. An extensive bronchiolitis is frequently a more serious disease than a bronchopneumonia of limited area. The existence of a bronchopneumonia in bronchiolitis, occurring simultaneously with it or usually after some time, seems probable by the increase of the existing fever, dyspnœa, and especially from the fact that the cough is painful and suppressed and expiration moaning and interrupted. Usually, however, one or two days elapse from the time when it is accepted that a pneumonia has set in until the corresponding physical signs appear in the lungs. At first fine metallic râles are heard in some particular spot, usually low down posteriorly, or the percussion note shows a slightly tympanitic accessory note. Soon, or perhaps only after some days, bronchophony,

bronchial breathing, and dulness (on light percussion), make their appearance. These signs, however, may also remain absent.

If the bronchiolitis becomes more and more general, or larger and more numerous, pneumonic areas develop, and a grave clinical picture is presented either very quickly or after one or two weeks. The frequency of respiration rises to 70–100; the respirations are superficial; and at times somewhat irregular. All accessory muscles assist laboriously during inspiration, even the lower jaw being drawn downwards. The inspiratory recessions of the thorax are of a high grade; the cyanosis increases in an alarming manner; and cold perspiration and apathy appear. The dyspnœa causes all efforts to take nourishment to be a torment. The infants lies with lustreless, half-closed eyes, and the sad look seeking for aid is directed towards the mother with head frequently drawn backwards. The pulse becomes very small, scarcely perceptible, 200–240 per minute, and the extremities become cold and swollen. Off and on, the infant still attempts to sit up only to fall back again into the pillows, exhausted. The previously harassing cough ceases, and the suppressed crying gives place to strengthless moaning. The apathy gradually develops into stupor and the increased cyanosis gives place to an ominous pallor. With increasing hyper-accumulations of mucus even in the larger air-passages, the little sufferer slumbers to a death that may often be preceded by slight convulsions.

If, on the other hand, improvement takes place, the respirations gradually become slower and deeper, and inspiratory recessions and cyanosis abate with gradual disappearance of dulness, bronchial breathing, and râles. Sleep and appetite are improved, and the pale, but no longer cyanotic features are once more enlivened by a smile. Recovery in bronchopneumonia takes place, on the average, after the second or fourth week, but frequently, when there are many improvements and relapses, only after some months. Even then relapses, diarrhœa, exhaustion, and other complications, may bring about a fatal termination.

Individual Symptoms. Seat and Spread of the Disease.—Bronchiolitis usually attacks both lungs simultaneously in larger areas, chiefly the posterior lower portions. Consequently, bronchopneumonia occurs bilaterally, and with predilection first or, indeed, only for the posterior inferior parts. From here the infiltration gradually spreads upwards as far as the middle of the scapula, becoming more and more distinct, but always remaining most distinct below, forming in this manner a stripelike zone which is therefore designated "*Stripe*" pneumonia, or more aptly paravertebral pneumonia (Gregor). The disease is usually more pronounced on one side than on the other, but may also remain unilateral. The paravertebral type is found as a rule in infants during the first year in those who constantly lie on their backs,

or at least the greater part of the time, a significant reason why bronchopneumonia favors so much the dependent and illy ventilated portion of the lungs. Laterally, the consolidation does not usually extend beyond the axillary line. Bronchopneumonia may, however, appear in any of the other portions of the lungs, in the upper lobes, and is very apt indeed to affect the small portion of the left upper lobe covering the pericardium.

Considerable experience in *physical examination* is required to demonstrate a beginning bronchopneumonia, and even in its subsequent stages well-marked symptoms of consolidation are usually shown much earlier by auscultation than by percussion. As an early sign of bronchopneumonia, in addition to medium and fine ronchi, there may be heard over a circumscribed area, usually below and behind, aggregated, fine, metallic râles. Some time later, or perhaps at the same time, the percussion note over this region becomes tympanitic. This is a very important sign, and is produced from the fact that the areas being about the size of hazel-nuts are not sufficient to diminish the resonance but may, however, produce a retraction of the intervening parts. It is characteristic to have the metallic râles first disappear again in one situation in order to reappear in another. If consolidation spreads and the areas become larger and more confluent, distinct bronchophony is heard on crying or speaking. Bronchial breathing and dulness appear. Should the left lower lobe contain larger areas, the heart's action may be heard through them more distinctly than normally. Whenever respiration is superficial, in young children the respiratory murmur is either diminished or increased with ronchi; while during speech, and especially while crying, distinct bronchophony and bronchial breathing are present. For this reason, auscultation of the infant while crying is always of especial value; and it is justifiable in doubtful cases to cause the infant to cry, during which bronchophony may be looked for. This has the same significance as bronchial breathing, and may be heard more frequently in children. Percussion must be very light, otherwise the loud resonance of the sound lung tissue will not permit dulness to become perceptible which is produced by smaller areas. Should extensive continuous consolidation occur, the percussion note is very much diminished and no longer yields the tympanitic accessory note; the finger receives the sensation of marked resistance while percussing, which is, however, not so great as in pleuritic exudates. Frequently, in bronchiolitis and especially in bronchopneumonia, characteristic distention of the uninvolved anterior portions of the lungs occurs (acute emphysema), especially along the borders of the same. This manifests itself by the depth of the lower pulmonary border on the right side anteriorly, and by diminution of the area of cardiac dulness. As recovery takes place the acute pulmonary distention subsides again. Vocal fremitus, which may also

be tested with the ear during crying, is frequently somewhat increased in extensive consolidation.

The *respiration*, which may be increased to 100 per minute, is superficial and at times irregular. The severe dyspnœa and the great inspiratory retraction of the diaphragmatic attachment and of the sternal notch with contemporaneous hoarseness may at the first glance simulate stenosis of the larynx. These recessions are especially marked in an existing rachitis of the thorax. The dyspnœa renders prolonged crying impossible, and also the holding of the breath which young infants usually do during auscultation. The nursling is frequently compelled to release the nipple. Very significant is the change in the ratio of respiration to that of the pulse, from the normal 1:3–4 to 1:2.5 or even 1:2. In bronchopneumonia expiration is moaning and cut short especially while crying, although not so marked as in croupous pneumonia. The cough is frequent and harassing, in bronchopneumonia often painful.

The *circulatory organs* are seriously involved. The pulse is very frequent, in severe illness of younger infants as high as 200, although this alone is not necessarily serious. Of far greater importance than the frequency of the pulse is the degree of arterial distention. From stasis in the pulmonary circulation the heart becomes over distended with blood, and, if of longer duration, dilated, which in whooping-cough may often be demonstrated clinically. Not uncommonly, fatty degeneration of the heart is found on section, rarely purulent pericarditis. Manifestations of stasis in the circulation (cyanosis, œdema of eyelids, hands, and feet) occur much earlier and more frequently in bronchopneumonia than in croupous pneumonia. Cases of sudden death are met with at times which are, however, less often due to cardiac failure than to rapid suffocation.

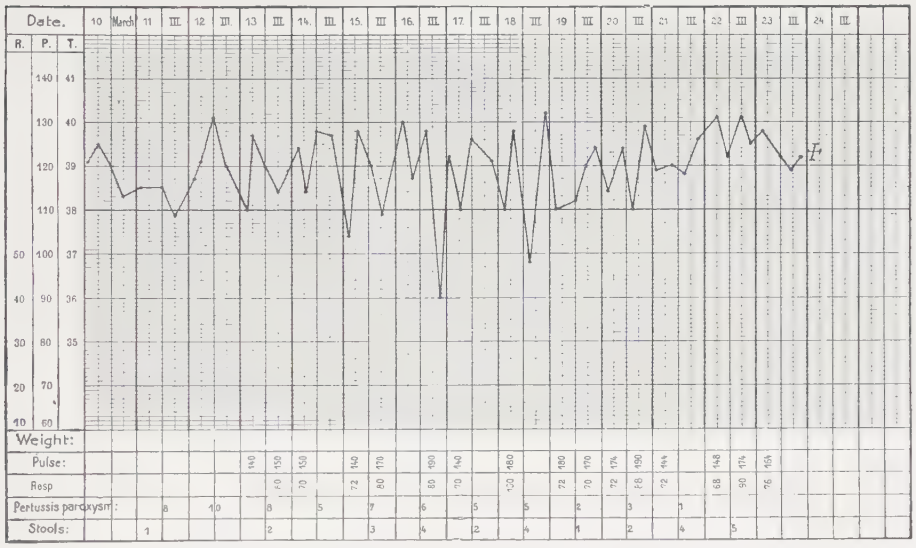
Organs of Digestion.—When the onset is more acute, vomiting often occurs. In nurslings, especially those who are rachitic, a troublesome meteorism is frequently present. During the first two years, diarrhœa and intestinal catarrh are frequent accompaniments of bronchopneumonia and are often responsible for a fatal termination. Occasionally, in protracted cases, enlargement of the liver and spleen occurs. The kidneys usually remain intact.

The **temperature** in acute bronchiolitis and bronchopneumonia is high in the beginning, and in favorable cases gradually returns to the normal. A gradual rise is frequently observed in cases slowly developing from a simple bronchitis. As a general rule, the temperature is not as high in bronchiolitis as in bronchopneumonia, so that a temperature remaining at 39.5° C. (103° F.) for any length of time often indicates a bronchopneumonia (Wyss). The temperature is, however, not typical in character. In bronchopneumonia it is mostly remittent, also intermittent, and shows great variations and sudden remissions, frequently

increasing from 40° to 40.5° C. (104° to 105° F.) (see Fig. 68). In cases of fresh involvement high elevations of temperature often occur. According to Comby, the pseudolobar form produces a regular temperature like the disseminated form. In weak and emaciated infants, the fever may often be entirely absent in bronchiolitis, as well as in bronchopneumonia (cachectic form). The temperature in chronic cases is also no criterion regarding the gravity of the case, and especially in fatal cases often shows a decline towards the end.

Special Forms of the Disease.—Not infrequently bronchiolitis rapidly terminates fatally in from 1–3 days, especially in young infants. There are cases of bronchiolitis with dangerous dyspnœa which show

FIG. 68.



Double bronchopneumonia after whooping-cough in a two-year-old rachitic child.

accentuated or absence of vesicular breathing without any fine râles. On section the large bronchi are found free, but, on the other hand, the finer bronchi are occluded with larger or smaller areas of atelectasis alongside (Henoch). In young infants, occasionally after an attack of coryza or false croup, there occurs a sudden threatening bronchiolitis (with few râles) which rapidly terminates favorably in two to four days. Henoch properly regards them to be of asthmatic origin. Perhaps, the acute bronchitis with congestion, described by Cadet de Gassicourt, also belongs to this class. Attacks of bronchiolitis in a circumscribed spot are occasionally found in chronic bronchitis and in pulmonary tuberculosis.

Several forms of bronchopneumonia are distinguished clinically:

1. A *disseminated form* in which small scattered areas are present without leading to extensive consolidation.

2. A *pseudolobar form* in which confluence of smaller areas, or even from the beginning a large portion or even the entire lobe, is involved. The mucus râles may be entirely absent so that the physical signs may correspond with those of a croupous pneumonia.

3. A *cachectic form*—frequent in feeble, rachitic children, suffering from gastro-intestinal diseases and progressing without, or almost without any fever. The frequent occurrence of bronchopneumonia in children suffering from gastro-intestinal diseases is regarded by some as a specific infection of the part through blood and lymph channels. The proof of this connection has never been submitted (Fischl, Spiegelberg).

4. A *protracted* and *chronic form* with a tendency to induration, pulmonary atrophy, and the formation of bronchiectasis.

Aspiration or *deglutition pneumonia* cannot be clinically separated from bronchopneumonia if abscess or gangrene of the lungs does not occur. Frequently pieces of food gain access to the lungs in feeble, stuporous individuals (meningitis), following tracheotomy in diphtheria of the pharynx and larynx, and acting as irritants set up an inflammation which may often lead to abscess or gangrene according to the bacteria which may be present. Henoch regards most pneumonias occurring in diphtheria as aspiration pneumonias. The septic pneumonias of the newborn result from aspiration of decomposed amniotic fluid (Silbermann). Probably, also the pneumonias of infants suffering from gastro-intestinal diseases are often aspiration pneumonias (Spiegelberg). The inflammatory changes affect the alveoli (necrosis of epithelium, distention with pus corpuscles) and inter-alveolar tissue (Plate 47). Small, gray, lobular areas are formed which often become necrotic. The enormous engorgement of the vessels of the bronchioles, characteristic of bronchopneumonia is absent (Aufrecht).

Secondary Pneumonia in Various Diseases.—Bronchopneumonia is the most frequent cause of death in whooping-cough and measles. In whooping-cough it is often dragging and more dangerous than in measles. In the latter instance, it often appears even during the stage of eruption, and may present great similarity with croupous pneumonia. It frequently progresses with a continuous high temperature, but does not terminate with the typical decline (Ziemssen). Bronchopneumonia occurring before the appearance of the exanthem is often rapidly fatal.

In epidemic grippe a genuine croupous pneumonia frequently occurs, often also a bronchopneumonia of the disseminated or pseudolobar variety, and sometimes even a mixed variety. In scarlet fever bronchopneumonia is not very frequent, but runs a severe course, and is apt to be followed by purulent pleuritis. Typhoid fever may be masked by an early bronchopneumonia. In rachitis it usually runs a very protracted course. In the newborn and during the first months of life the disease frequently produces neither fever nor marked cough and

very little cyanosis and dyspnœa, and is frequently recognized only after death (Miller).

Complications. — Mild dry pleurisy is frequently found, more rarely exudative, and then mostly purulent. Purulent arthritis and meningitis are rare. Of frequent occurrence and debilitating is the advent of *acute otitis media* (unnoticed in the beginning) which frequently leads to perforation and may be the cause of high temperature. Teichmann found otitis in 50 per cent. of his cases of bronchitis and pneumonia. Diarrhœa and intestinal catarrhs are apt to supervene, especially during the summer. Occasionally, during the course of a protracted bronchopneumonia, miliary tuberculosis is apt to occur. Formerly, a transition of bronchopneumonia to miliary tuberculosis was regarded as frequent. It is however decidedly rare (Ziemssen, Aufrecht).

The **prognosis** is always doubtful. The younger the child, the less is the chance for recovery. During the first year, more than one-half of the cases die. The prognosis is rendered decidedly more unfavorable by an active rachitis, general debility, gastro-intestinal disturbance, and insufficient care. Even in the course of the disease itself, rapid changes for better or worse occur. Capillary bronchitis may even on the first or second day, before the parents think of sending for a physician, lead to sudden death from suffocation. In ordinary cases the prognosis depends on the degree of dyspnœa, the inspiratory recessions, the cyanosis, and the quality of the pulse.

The **diagnosis** of capillary bronchitis is made from the scattered medium and fine subcrepitant râles; that of bronchopneumonia from appearance of smaller or larger areas of consolidation which lead to diminution of the percussion note, to bronchial breathing and also to circumscribed metallic râles, undetermined breathing, and slight tympanitic pulmonary resonance. Mostly, there is doubt whether a capillary bronchitis or a bronchopneumonia is present. Frequent careful examination and observation of the above-described symptoms usually lead to a proper differentiation in the course of a few days.

We do not consider it justifiable towards the attendants to make a diagnosis of pneumonia on theoretical grounds, so long as this cannot be made from the result of clinical examination. Acute miliary tuberculosis may produce similar symptoms to bronchiolitis; and in cases of older children this possibility must be kept in mind. Bronchopneumonia differs from other pulmonary affections often more by its origin and by its course than by its physical symptoms.

It is often difficult to differentiate the pseudolobar form from croupous pneumonia in so far as the early course is not definitely known, and only a few râles limited to the consolidation are present. In favor of croupous pneumonia are the sudden onset in the midst of perfect health, the high, continuous temperature, rapid development of dulness,

and critical decline of temperature. Gradual development, bilateral occurrence, the seat of disease at the inferior posterior portion of the lower lobe, severe dyspnœa, and cyanosis point to bronchopneumonia, as well as often the etiology (measles, etc.). Occasionally, in influenza there is added to an extensive bronchitis a genuine croupous pneumonia. In this instance only the subsequent course of the disease will decide the nature of the pneumonia. At times, great and often for a long time insuperable, difficulty is presented in the differentiation from acute pulmonary tuberculosis, especially in the form of a caseous pneumonia which sometimes develops in the lower lobe in children. The physical finding is exactly the same as in bronchopneumonia. Developing during the course of measles, whooping-cough, high grade rachitis, with patients less than three years old, indicate bronchopneumonia. In suspicious cases we are more likely to be dealing with a bronchopneumonia than with the more infrequent tuberculosis. Slight pleuritic exudation (or adhesions) in the lower inferior portions with an accompanying catarrh may give rise to confusion. The increased sensation of resistance, the diminished fremitus, point to pleurisy and speak against pneumonia. The differentiation of bronchopneumonia from hypostasis or extensive atelectasis is very often found to be impossible and practically is of no great importance.

FIG. 69.

Bronchitis croup kettle.

The **prophylaxis** coincides with that of simple bronchitis (page 334). It is important, but difficult to execute, to keep away from infants, all persons affected with colds and sore throats.

If it is possible to postpone infection of measles and whooping-cough until the third or fourth year, much is done towards the prevention of bronchiolitis and bronchopneumonia. Catarrhs in young infants must be carefully treated. Every harmless coryza in a delicate or rachitic infant may lead to a fatal bronchopneumonia. Small children who are bedridden must be guardedly carried about.

The **treatment** may affect the course of the disease to a great extent, and by rational methods may often act in a life-saving manner. As soon

as a serious bronchitis occurs in a young infant, the best and sunniest room in the house should be set apart, all superfluous furniture removed, and all unnecessary persons and visitors kept at a distance. Of the greatest advantage, especially during the cold season of the year are two connecting rooms for alternate use, the vacant one being thoroughly aired and cleaned during the intervals. Provision should be made for frequent changes of air, and the temperature kept at 15°–16° R. (66°–68° F.). The air must be kept moist. This is best accomplished by means of the croup kettle at the bedside, the vapor of which is directed towards the patient. The bed must be free, and the head should not be buried in soft pillows. Small infants must be carefully carried about; and older children must frequently change their position. The infant should frequently be laid for one half hour on its abdomen for a better aëration of the posterior portions of the lungs.

The greatest care must be given to the *nutrition*. On account of the average long duration it is of importance to nourish the organism well from the start, and to avoid everything which might disturb the appetite and digestion. The diet in acute cases must be a fluid one, and should consist chiefly of milk, broths, gruels, etc. There is less likelihood of diarrhœa and intestinal catarrh if the milk is diluted (with water, rice or barley water) from the start, in children from three to four years of age. For the purpose of supporting the nutrition small doses of enterorose, plasmon, nutrose, etc., are of advantage. Excellent results are obtained during the second year from the expressed juice of fresh beef. Drinks are to be offered freely, the best being boiled sugar water (with a tendency to diarrhœa—milk-sugar). Ems water readily causes diarrhœa. Teas are usually well borne if the tea is freshly prepared and not allowed to stand on the stove for hours. Disturbances of digestion must immediately be combated by changes in the diet and ultimately the withdrawal of causative drugs. *Good digestion* is of *greater importance* than expectorants. Care of the mouth and skin must be closely supervised; and the ears, on account of the frequent and unobtrusive occurrence of otitis media, should be examined from time to time.

The **treatment** of *bronchiolitis* and *bronchopneumonia* is practically the same. Of the numerous remedies which are employed I regard the chest compress, baths, stimulants, inhalations of oxygen, and thorough moistening of the air, as the most important. In acute cases, both for the pulmonary symptoms and the temperature, wet compresses and lukewarm baths in the beginning prove most efficacious. In cases of high temperature, chest compresses (water at the temperature of room) are applied every 1–2 hours during the day; in older children with a very high temperature they are changed every half hour or even every quarter hour, the water being at a temperature of 12° R. (64° F.). In severe

cases compresses are also employed during the night. In addition a lukewarm bath of 27°–25° R. (93°–89° F.) is given morning and evening for 6–10 minutes. The skin, especially the arms and legs, is to be rubbed during the bath, the forehead and face sponged with cold water. The physician must, at least at first, supervise the application of the compresses and baths and satisfy himself as to the proper application and action. Frequently, mostly in nurslings, discomfort, cold skin, and cyanosis occur after the use of the compresses. In this case, the compresses are dispensed with and only warm baths of from 28°–30° R. (98°–100° F.) given. Koplik even recommends baths of from 32°–33° R. (105°–106° F.). Hot baths of 15 minutes duration twice daily from the onset of the disease are praised by Renault, who claims that the pulmonary congestion is reduced thereby and a favorable action obtained on the heart and temperature.

In cases where the fever is mild, or absent, warm compresses about the chest and covering them with rubber are, according to my experience, of utility, as well as often in protracted febrile forms. In acute febrile forms, and in delicate and young infants, the warm compresses as recommended by Jürgensen, often give better results than the cold. In addition to these, the cool skin is warmed by dry frictions, and the thorax is rapidly sponged with cold water every time the compresses are changed, thus producing deep inspirations.

I have frequently convinced myself that the routine application of cold compresses and cool baths in delicate and weakened individuals has lead to cyanosis, acceleration of pulse, cold extremities, etc., while hot baths of from 30°–32° R. (100°–102° F.), even when fever is present, have apparently acted well and quieted the patient.*

Besides the baths and compresses, the *fever* requires no direct treatment. Frequently cold applications to the head and cold spongings of the extremities followed by dry friction are agreeable. If a febrile remedy is desirable quinine may be given in small doses per rectum. (Quinine muriat 1.0 Gm. (15 gr.) acid muriat. dil. q.s. ad. solut., aq. destill. ad 100.0 c.c. (3 oz.) for three or four enemata).

With the onset of numerous râles, expectoration must be vigorously stimulated. Liq. ammon. anisat. (P. G.) 2–8 drops three to five times may be given, or elix. e. succo liquirit. (P. G.) 10–40 drops three to five times. Where there is considerable accumulation of mucus, a decoction of senega root acts more energetically.

Decoct rad. senegæ.....2.0–6:50.....gr. xxx–℥iss to ℥ii
Liq. ammon. anisat.....1.0–2.5.....gr. xv–gr. xxxviii
5 Gm. (1¼ dr.) every 2 hours.

* Baelz in Tokio regards the hot bath in capillary bronchitis as being almost a specific, acting as a derivative to the entire skin and improving sleep, fever, cough, and pulse. A bath of from 40°–42° C. (104°–106° F.) is repeated whenever necessary and continued for 5–15 minutes. If the temperature is very high the face is sprinkled with cold water and the body is immersed only as far as the nipples. When the temperature is moderate a bath of longer duration is given.

In cases of failure of respiration and weakness, camphor with benzoin is to be given (camphoræ trit. 0.01–0.03 Gm. ($\frac{1}{6}$–$\frac{3}{4}$ gr.) with flor benzoes 0.01 Gm. ($\frac{1}{6}$ gr.), every one to three hours. In cases of disturbances of digestion all these remedies had better be omitted, and camphor or caffeine be given subcutaneously. Caffeine natrobenzoic 1.0 Gm. (15 gr.) aqua destill. 10.0 Gm. (3 dr.), $\frac{1}{4}$ to 1 syringeful two or three times daily. Narcotics often act directly very injuriously by diminishing the expectoration.

For the purpose of emptying the bronchi thoroughly an emetic is recommended by some in the beginning of a violent capillary bronchitis. It is best to give ipecac in full doses, but not when the stomach is empty, *i.e.*, rad. ipecac pulv. 0.5 (8 gr.) in syrup. Should vomiting not take place, the same dose is repeated after ten minutes, and if necessary again in another ten minutes. Vomiting is hastened by allowing the patient to drink plenty of water.

According to my personal experience, I have never seen any particular good results from the use of emetics. At any rate an emetic may only be given to a robust infant and in the beginning of the illness. If weakening and carbonic acid poisoning are already present, vomiting will not take place, but more likely a serious collapse.

In very young infants, Schultze's method also acts well by emptying the bronchi (Engel).

Although *mustard baths* have long been in use, recently Heubner very warmly recommends a more energetic method of application in the shape of a mustard pack. The procedure may be adopted as soon as a diagnosis of bronchiolitis has been made.

About 3 tablespoons of fresh mustard flour are stirred in $1\frac{1}{2}$ litres (3 pints) of warm water (104° F.) for about 10–15 minutes until the odor becomes perceptible from the development of the mustard oil. A cloth is now dipped in this mustard mixture, wrung out well, and the patient enveloped from the neck to the feet, and wrapped in a woolen blanket. The head only is allowed to remain free. The neck is still more carefully covered so that the pungent odor of the mustard vapor does not irritate the lungs. The patient is allowed to remain in this pack for 10–20–30 minutes until he is as "red as a lobster," after which the patient is removed from the pack, cleansed with lukewarm water, or given a lukewarm bath at 28° R. (98° F.), and kept from one and a half to two hours in an ordinary long moist pack so that the hyperæmia of the skin may be maintained as long as possible. Afterwards the patient is again bathed and if necessary is given a cold sponge, dried, and allowed to lie quietly. The hyperæmia of the skin which has been attained (which probably acts by depletion of the lungs) often lasts for several days. The procedure is carried out not more than once a day. I have also several times seen surprisingly good results from the use of these energetic mustard packs in acute cases.

In cases where the breathing is very superficial, with beginning of carbonic acid poisoning, cold irrigations during a warm bath act beneficially by increasing the depth of the respiratory movements.

The patient receives a bath of from 28° to 30° R. (98°–100° F.) three or four times daily, during which the skin is vigorously rubbed until it becomes red, and at intervals of about twenty seconds a stream of cold water (usually from a small sprinkling can) is poured down the nape of the neck for five or ten seconds. A heart stimulant is to be given just before the irrigation. Where there is marked prostration the cold irrigation had better be omitted on account of the danger of collapse. Slapping the back vigorously with cold wet cloths stimulates the respiration.

Fig. 70.

Inhalation of oxygen. Oxygen cylinder with regulating manometer.

Inhalations of oxygen are useful in cases of severe dyspnœa as soon as the cyanosis indicates the decarbonization of the blood is insufficient. They are especially recommended by Hagenbach-Burckhardt, and when used freely and frequently give very good results as long as many bronchioles are still patent and no extensive bronchopneumonia is present; that is, if the cyanosis is dependent principally on a stenosis of the air-passages (bronchiolitis) and not on an encroachment of the respiratory surface. The patient is simply allowed to inhale the oxygen through a glass funnel loosely placed over the nose and mouth (see Fig. 70).

Good results are reported from venesection in some cases of high grade cyanosis with sufficient strength. About one tenth of the total quantity of blood or about one one-hundred-and-thirtieth of the body's weight of blood is removed (Heubner).

Stimulants should be given early. Beef tea, bouillon, tea, and coffee, as an addition to milk, are to be considered first. A stimulant should always be given before the application of the cold packs, baths, and especially before cold irrigations. In mild cases these dietetic

stimulants are sufficient. One should be cautious regarding the use of alcohol. Caffeine is more to be recommended in cases of weakening pulse (Caffeine natrobenzoic 0.2–1.0 Gm.: 100.0 c.c. (2–15 gr. to 3½ oz.) 5 Gm. (1¼ dr.) 3–6 times daily in sugar water). In protracted illness, digitalis often acts well (very convenient in the form of digitaline Homolle et Quevenne, 1 granule a day).

In cases of increasing weakness, camphor and caffeine are given subcutaneously. Koplik recommends a daily rectal injection of warm normal salt solution as beneficial to the heart's action; likewise, normal salt solution may be injected subcutaneously. Strychnine is much praised by English and French physicians.

During *convalescence* the patients may be taken out of doors during the summer after the disappearance of the fever, even if some slight signs of consolidation are still present. As a tonic, quinine and in cases of marked anæmia iron (bioferrin) and in rachitis phosphorus are useful. Later removal to the country is excellent. As an after-cure the sea-shore or mountains are to be recommended, and in cases of remaining catarrhal conditions, a sojourn during the winter in a warmer climate.

BRONCHIAL ASTHMA AND ASTHMATIC BRONCHITIS

Occurrence and Etiology.—From the vague general conception of "Asthma," we may definitely separate bronchial asthma. The same is characterized by the occurrence of conditions of expiratory dyspnœa, pulmonary emphysema with dry bronchial catarrh, and is dependent on a neurosis of the respiratory system which is to be regarded mainly as a spasm of the muscles of respiration, or perhaps also as a neurosis of secretion of the mucous membrane of the organs of respiration.

The pure bronchial asthma with intervals of a normal respiratory system is seldom found in children; it is, however, met with occasionally in infancy, typical cases of which I have observed. On the other hand, during the whole of childhood from the first year on mixed forms of bronchitis which may best be designated as asthmatic bronchitis are very frequent.

Hereditary circumstances have considerable influence. Bronchial asthma very frequently attacks children whose parents likewise suffer or have suffered from asthma, gout, migraine, or are otherwise affected neuropathically. Accordingly, neuroarthritismus plays an important rôle in this condition. Asthma itself is mostly developed from peripheral irritation, from chronic nasopharyngeal affections, especially from adenoid vegetations; and also, from chronic relapsing bronchitis. Eczema is important, and, less so, other affections of the skin, as strophulus infantum, and chronic urticaria. The French speak of a *diathese dartreuse* and of *asthma dartreux*. It seems to me that the connection between eczema is not, in general, sufficiently appreciated in German

literature. According to my own personal experience, I have found that the vast majority of cases of bronchial asthma or asthmatic bronchitis occur in children who have suffered from, or are still suffering from, infantile eczema. Certainly, in addition, adenoid vegetations are also frequently present. The first manifestations very frequently appear during the second year, when the eczema has healed or is about to disappear, a clinical fact which not improperly permits the layman to speak of "a striking in." Children who are predisposed to asthma are often anæmic, nervous, and irritable. Changes of weather and climate, certain odors, fresh colds, and psychic factors, may be regarded as exciting causes. In certain children every attack of bronchitis assumes an asthmatic type.

Hay asthma is a type of bronchial asthma which is produced by irritation of the nasal and deeper seated respiratory mucous membrane, from the pollen of various plants especially of grass and grain. The predisposition to hay fever is hereditary and is found in family neuro-arthritismus. The disease sometimes commences at three to six years as a hay-cold, and frequently remains unrecognized during the first years, if the conjunctivitis and the well-known predisposition do not lead to the proper diagnosis. Pronounced hay asthma mostly occurs several years later. However I have seen a severe attack of pure hay asthma in a boy only four years old, of a family in whom no symptoms of any kind of the disease had been observed during previous years.

The **symptoms** of bronchial asthma are as marked in children as in adults. After a short indisposition severe dyspnœa suddenly sets in; filled with anxiety and with pale features the infant attempts to cling to anyone. Laborious, prolonged, and panting expiration is present, whereby all the auxiliary muscles are strained and the active abdominal muscles in particular are severely taxed. The thorax itself is rigid or makes but slight excursions and inspiratory epigastric recessions occur; cyanosis, often also cold sweats, sets in. During the early years of life, spasm of the glottis may be added. Respiration is frequently retarded. In addition to the panting expiration, dry, sibilant râles perceptible over the entire room often occur early or occasionally only after some time.

Auscultation reveals diminished respiratory murmur, and in addition very frequently the numerous sibilant and sonorous, mostly expiratory ronchi just mentioned are heard over the entire lung. Percussion yields a loud hollow note and marked emphysema of the lungs, the border of which may extend to the eighth rib on the right side anteriorly. The pulse is small and unusually accelerated. The temperature, on the other hand, is normal in pure bronchial asthma. During sleep, the dyspnœa abates. Cough is frequently absent in the beginning; but, on the other hand, sets in towards the close of the attack when the ronchi

become somewhat more loosened, bringing to light in older children a tough, purely mucus expectoration which contains Charcot-Leyden crystals, Curschmann's spirals, and many eosinophile cells. The attack lasts for several hours, but may also continue for days and exhaust the patient very much. Towards the close of the attack the dyspnœa rapidly subsides, emphysema and ronchi disappear. After weeks and months recurrences of the attacks frequently take place. The evil generally lasts for years.

As already mentioned above, *asthmatic bronchitis* in children is much more frequently met with than pure bronchial asthma. There are individuals in whom every new attack of bronchitis (often febrile) immediately assumes an asthmatic character, *i.e.*, it begins with sonorous ronchi, moderate emphysema, and increased expiration. These asthmatic symptoms then gradually disappear with the resolution of the dry catarrh, and the usual remaining bronchitis gets well in a few days or weeks. These children suffer from this kind of bronchitis once or twice a year; and also in the intervals of freedom they are often somewhat short of breath and frequently have chronic nasal catarrh and adenoids.

The **diagnosis** is based on the acute pulmonary emphysema and the expiratory dyspnœa. A number of types of asthma originating from a nervous, hysterical, uremic, dyspeptic (asthma dyspepticum), basis are thereby eliminated from the beginning, as well as a number of diseases which predominantly cause inspiratory dyspnœa, such as spasm of the glottis, paralysis of the recurrent laryngeal nerves, stenosis of the trachea, foreign bodies. Enlarged bronchial glands may also produce attacks of dyspnœa. *Cardiac asthma* causes no pulmonary emphysema; inspiration and expiration being equally dyspnœic. If the bronchial asthma is accompanied by catarrhal laryngitis, the clinical picture will show similarity with genuine laryngeal croup in which, however, inspiration is predominantly interfered with, the stenosis is more marked and develops gradually, and pulmonary emphysema and sibilant râles are absent. The disease in very young infants is more apt to simulate *capillary bronchitis*. Dyspnœa on inspiration and expiration, marked inspiratory recessions, fever, moist, fine mucous râles from the beginning, speak in favor of capillary bronchitis.

The **prognosis** in individual cases is good. The predisposition to bronchial asthma may, however, remain during the whole lifetime and lead to the development of true emphysema. Frequently, on the other hand, towards puberty there may be an abatement and disappearance of the attacks.

Prophylaxis consists in the removal of adenoid vegetations, careful treatment of nasal and bronchial catarrhs, and eczema, strengthening and hardening of the system, much outdoor exercise, prolonged sojourn in the country or mountains, removal from large cities, curtailing of

animal food, avoidance of overfeeding, abundant supply of vegetables and fruit. Prolonged milk diet and arsenical treatment are at times of utility.

The **treatment** has for its first object the relief of the attack, for which chloral hydrate (frequently 0.25–0.5 Gm. (3–7 gr.) in enema) seems to be the most suitable. Also, codeine, or morphine, in older children is useful. For the relief of the dry catarrh, potassium iodide or sodium iodide (1.0–5.0 Gm. (15–75 gr.) succ. liquirit 3.0–5.0: 100.0 (45–75 gr. to 3½ oz.) 5 Gm. (75 gr.) 5 times daily, not to be repeated) is of value. In cases which terminate tardily, ammon. bromat. 1.0–5.0 Gm.: 100 c.c. (15–75 gr. to 3½ oz.) may be tried, with warm vapors, and the further treatment of ordinary bronchitis. In frequently recurring asthma, systematic gymnastics of the lungs is often useful. In hay asthma, Pollantin sometimes gives good results.

BRONCHIECTASIS

Occurrence and Etiology.—Slight dilatations of the finer bronchi may rapidly occur even in acute bronchial catarrh, especially in the course of whooping-cough, but they may also disappear very rapidly. Chronic bronchiectasis, which may be clinically diagnosticated, occurs congenitally in rare instances, and at times develops very slowly in older children, mostly following acute diseases of the pleura and lungs, and typical pneumonias terminating in chronic induration, bronchopneumonia of measles, whooping-cough, or influenza. It also follows pleurisy which has led to thickening and adhesions, and stenoses (foreign bodies, syphilis). The dilatation of the bronchi develops from expiratory pressure (in bronchitis), or from inspiratory efforts (pulmonary and pleuritic shrinkings).

Pathological Anatomy.—The dilatations are cylindrical, spindle-shaped or sacculated, diffuse or circumscribed. Clinically, the circumscribed, sacculated dilatations, which are chiefly found in one of the lower lobes and proceed from a medium sized bronchus, are principally to be considered. Beside these smaller cavities are often found. The mucous membrane is atrophic in advanced cases, elastic fibres, muscles, and even cartilage, having disappeared. The cylindrical epithelium is replaced by pavement epithelium, or is even destroyed by granulations and ulcerations. The mucopurulent contents of cavities which may be as large as hen's eggs, show an enormous number of bacteria (strepto- staphylo- pneumococci, and many anaërobia).

Symptoms.—Numerous obstinate ronchi always in the same situation (usually in the lower lobe) in the course of a chronic bronchitis lead us to surmise the presence of a larger dilatation; and this is strengthened by the offensive odor of the abundant expectoration. Frequently, months and years elapse until the manifestations become plainer, especially as long as the patients swallow the expectoration.

The *cough* is often characteristic in so far as the general bronchitis which usually accompanies it does not influence it to any material degree. Frequently, the patients do not cough for hours and are then harassed by violent paroxysms of coughing which occur after a change of position, such as lying over on the sound side on awakening in the morning. They then cough up a very abundant secretion which is thin, liquid, grayish yellow, mucopurulent and which in standing separates into the well known three layers. The expectoration is often somewhat offensive, though usually not so strong as in fetid bronchitis, at any rate not so stinking as in gangrene of the lungs. The expectoration occurring in gushes and "mouthfuls" is a striking condition, which occurs only in rupture of an empyema into the bronchi. The expectoration is frequently bloody, and severe hæmoptysis may also occur.

Only in larger cavities and peripheral situations are the local symptoms unequivocal. Numerous coarse râles are then heard over a circumscribed spot, which at times, especially after coughing, produce a gurgling sound. In the same situation there is often bronchial breathing and bronchophony. The percussion note is tympanitic, sometimes also diminished because the bronchiectasis is frequently surrounded by thickened lung tissue which causes the râles to be heard more distinctly. True cavernous signs are rare in children. The change in the symptoms according to whether the cavity is filled with secretion or is empty is pathognomonic, as are also the disappearance and the reappearance of the tympanitic sound and dulness, bronchial breathing, etc. The accumulation and stagnation of the secretion often cause irregular elevations of temperature and fetid breath. Repeated occurrence of small pneumonic areas always in the same spot often indicates a bronchiectasis. In addition to the commonly accompanying diffuse bronchitis, a pleurisy, generally purulent, often occurs. Additional symptoms, such as dyspnœa, clubbed fingers, pulmonary emphysema, pleuritic thickening, chronic pneumonia, displacement of the heart, etc., are often connected with the primary disease. The course frequently extends over many years with varying improvements and relapses. Additional complications of pulmonary gangrene, bronchopneumonia, metastatic abscesses, amyloid degeneration, tuberculosis, which may furnish the cause of death, may be mentioned, besides anæmia and exhaustion with hectic fever.

The **diagnosis** is made from the above described symptoms, of which the persistent râles over a circumscribed spot, periodical attacks of coughing with enormous expectoration, besides a tympanitic percussion note, and bronchial breathing over a circumscribed area, are the most important. Fetid bronchitis often follows a similar course, but is without any special local symptoms. The course of gangrene of the lungs is much more acute and serious, the expectoration containing pieces of discolored parenchymatous tissue, the breath having the usual characteristic

odor. Abscess of the lungs yields a pure purulent expectoration without offensive odor. It is often impossible to distinguish it from a localized emphysema with which it may be confounded, even after exploratory puncture (Koplik) or after rupture through a bronchus. In contrast to tuberculous cavities (rarely large in children) there is in bronchiectasis less disturbance of the general health, and an absence of tubercle bacilli in the expectoration. Thickenings and adhesions are also in favor of bronchiectasis.

The **prognosis** is on the whole bad. The disease may be stationary for years, but recovery is rare. Dangerous complications always threaten, and the fatal termination is mostly in the form of pseudophthisis.

The **treatment** is directed to increasing the strength of the patient, combating the anæmia with nourishing food, and whenever possible with out-of-door life in the country, mountains, sea-shore, and in the south during the winter.

In addition to the occasionally administered expectorant (liq. ammon. anisat.) for the purpose of promoting the expectoration, beside cough-producing positions, systematic expiratory compression of the thorax (Gerhardt) should be practiced. To limit the amount of secretion, inhalations of ol. terebinthin., ol. pini pumilion., aq. picis, ferric chloride $\frac{1}{2}$ per cent.; internally, creosote, guaiacol carbonate, terpin hydrate, balsams, are to be recommended. Vierordt has recently obtained very encouraging results from mobilization of the thorax (multiple resection of ribs). Formerly, the results of operative interference were unfavorable (Tuffier).

STENOSIS OF THE TRACHEA AND OF THE LARGE BRONCHI

A narrowing of the trachea or of the bronchi occurs, in rare cases (Gregor), congenitally, otherwise almost always only as a symptom of various diseases. A short description is therefore given here for purely practical reasons only. The origin of the stenosis frequently lies in disease of the air-passages themselves. The most important cause is the formation of membrane in diphtheria, more rarely in fibrinous bronchitis, then also formation of granulations and softening and defects of the trachea after tracheotomy, syphilitic ulcerations and scars, foreign bodies, etc. In the second place, pressure from neighboring structures may lead to compression stenosis, as struma (often substernal) enlarged mediastinal and bronchial glands, burrowing abscesses, pleuritic adhesions, hygromatous cysts. Hypertrophy of the thymus has also been seen by me to be a cause of chronic stenosis of the trachea.

The **symptoms** come on either rapidly (foreign bodies) or slowly according to the cause. Respiration is more or less retarded and laborious, especially inspiration which, during circumscribed stenosis, is often accompanied by a whistling and panting noise (stenosis sound). In the

sternal notch, but more distinctly in the epigastrium, inspiratory recessions appear, especially in narrowing of the trachea, to which severe cyanosis may also be added. If the cause lies in enlarged mediastinal glands, in addition to the loud inspiratory stridor, attacks of panting and wheezing cough of a high note frequently occur. Swelling of the bronchial glands producing stenosis may aften cause slight exophthalmos (Friedjung). Percussion shows a normal condition, whereas in characteristic contrast thereto, the fremitus and vesicular breathing of the affected portion of the lung is diminished or absent.

Diagnosis of Seat.—A clear voice and the moderate excursions of the larynx indicate that the stenosis is farther below. In tracheal stenosis the stenosis sound is usually more marked, dyspnœa more threatening, as if the obstruction affected only one large bronchus. In addition the cause is often directly shown (struma, scars resulting from tracheotomy). If the stenosis is in a bronchus, vesicular breathing, fremitus, and movements of the thorax, are diminished on that side only, while the free portion of the lung is often excessively stretched and appears distended. The treatment is described in the respective chapters.

CONGENITAL STRIDOR (STRIDOR CONGENITUS)

By the terms, congenital stridor, stridor congenitus, or stridor neonatorium, is meant a rare peculiarity of respiration which may occur immediately or soon after birth. Inspiration is attended by a loud or cackling noise which in increased respiration may also remind one of the sound emitted by a rooster or an excited hen (Thomson). At the same time, inspiration is accompanied by a recession of the jugulum and of the epigastrium. Expiration is mostly without any noise. Cyanosis is absent. The general health remains unimpaired. This peculiar inspiration often increases in frequency until the middle of the first year, then gradually diminishes, and usually disappears in the course of the second year. The nasopharyngeal space is normal.

The **etiology** is obscure and disputed. In some of the cases, an anatomical change in the epiglottis has been pointed out, first by Lees in 1882. The epiglottis is folded in such a manner that the lateral edges touch each other and allow only a small slit of the aryepiglotteal folds, which is still more contracted by the draught of the inspired air. Avellis, Hochsinger, and others, regard congenital stridor as a slight stenosis of the trachea, the cause of which they hold to be an hypertrophic thymus, which Hochsinger believes can sometimes be demonstrated by radiography.

By *struma aërea* (air-goitre) is meant a very rarely observed cystic tumor filled with air in the anterior lateral region of the neck, which communicates with the larynx, the trachea (tracheocele), or with the bronchi. The air-goitre develops spontaneously or traumatically, and

has been seen in small children even during the first year of life. The air-cyst usually remains small, is soft, disappears (and sometimes crackles) on pressure, and enlarges during crying. It is distinguished by its circumscribed shape from subcutaneous emphysema.

FOREIGN BODIES IN THE TRACHEA AND IN THE BRONCHI

Foreign bodies not infrequently find their way into the trachea and into the bronchi in children while eating (coarse bread crumbs, small pieces of bone), or while playing (buttons, nails, pebbles, beans, corn, etc.), being aspirated while laughing or coughing. Frequently the foreign body lodges in the larynx and produces symptoms similar to croup. It may still after some time get into the trachea. Smooth, rounded objects often immediately glide into the trachea after an attack of suffocation, where they at once lodge or remain mobile according to their size and shape, or if of smaller circumference may slip into a bronchus.

If the foreign body which gains access to the trachea is large, rapid suffocation may result. Quite frequently, it remains movable and is then sometimes moved up and down with an audible clapping or fluttering noise, or it may be felt by the finger placed on the trachea. It produces a painful cough by which it is thrown against the glottis which closes spasmodically from its impact, bringing about at times a severe attack of suffocation. By a fortunate accident, occasionally, the foreign body may soon or perhaps, only after some time, be coughed out. If the foreign body becomes lodged anywhere it causes little inconvenience except local pain and symptoms of stenosis often without any particular symptoms of irritation.

Periods of rest and attacks of suffocation (in transitory mobilization) may alternate. Auscultation will yield, according to circumstances, either a negative finding, a flapping sound between the shoulder blades, or symptoms of stenosis of the trachea; while from injury or ulceration of the mucous membrane there is often a bloody or sanguinopurulent expectoration. Smaller objects sooner or later get into a main *bronchus*, and in the majority of cases (over two-thirds) into the right bronchus. When the main bronchus becomes entirely or to the greater part occluded characteristic signs are produced. In addition to the dyspnœa, which at first threatens, the respiratory movements, the respiratory murmur, and the fremitus, are diminished or abolished in the affected side. The percussion note is normal, except that there is lacking a displacement of the lower borders of the lungs. If a bronchus is only partly occluded, a loud whistling sound is heard over the corresponding side of the thorax.

In complete occlusion of a large bronchus the isolated portion of the lung rapidly becomes atelectatic. Moreover, symptoms of bronchitis and bronchopneumonia with fever set in usually after a few days; and in the same manner also in occlusion of a smaller bronchus. Subse-

quently, abscesses, bronchiectasis, pleurisy, pyopneumothorax, and more rarely gangrene of the lungs, frequently develop. The sound side shows compensating emphysema.

The **diagnosis** of the foreign body is usually easy if the history plainly points to it; otherwise, in the case of a tightly wedged body, it is often very difficult, unless one's attention is called to it by an initial suffocative attack or the appearance of acute symptoms of stenosis and bloody sputum. Circumscribed bronchopneumonia in robust children especially in the right lower lobe, which without further cause occurs acutely, must lead one to suspect a foreign body, especially if this bronchopneumonia, or bronchiectasis, recurs frequently in the same spot. The rupture of a caseous bronchial gland into a main bronchus often produces symptoms similar to a foreign body. The diagnosis is frequently rendered easy by an X-ray examination. Where nothing is known regarding the aspiration of a foreign body, or where the symptoms only appear after some time, the diagnosis is often never made and the patients die of pseudophthisis, bronchopneumonia, etc.

The **prognosis** is always very grave, as fatal suffocation and dangerous complications may set in at any time. The prognosis in cases of aspirated corn is very bad.

Treatment.—An attempt may be made to expel the foreign body by vomiting and retching; in cases of smooth heavy objects, suspension by the legs and slapping the back may also occasionally accomplish the purpose. But these procedures are not entirely free from danger. The most certain remedy is always tracheotomy as soon as the diagnosis is established. It is also to be recommended when the foreign body is freely movable in the wind-pipe and causes no serious disturbance, since one is never safe from sudden suffocation and incurable pulmonary changes. Tracheotomy is advantageously preceded by a preventive intubation. The foreign body is often coughed out spontaneously on opening the trachea, though frequently it must be removed with a suitable instrument, or wire snare. For this purpose Killian's bronchoscopy may give valuable service. After the successful removal of the foreign body, the tracheotomy wound is immediately closed. Foreign bodies in the second division of the bronchi cannot usually be reached by tracheotomy.

CROUPOUS (FIBRINOUS) PNEUMONIA

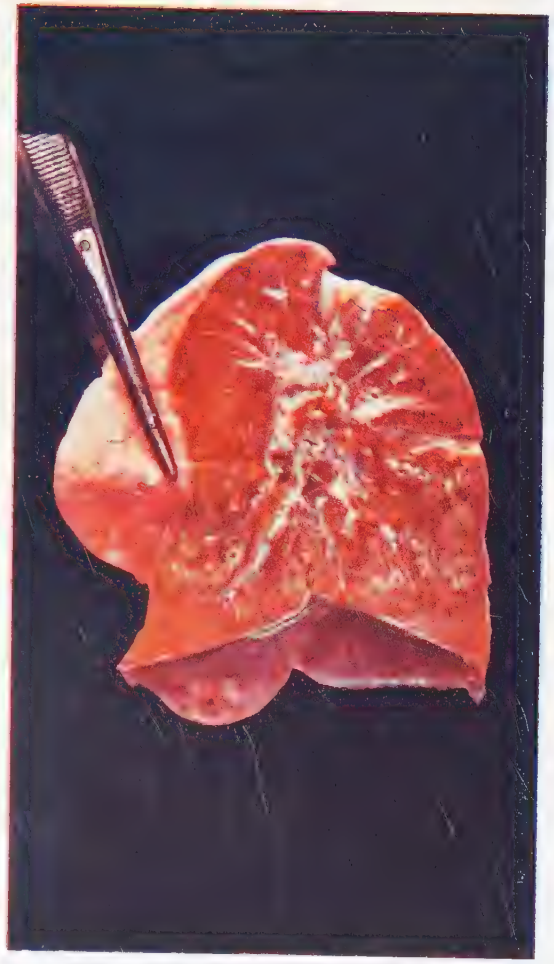
Croupous pneumonia constitutes a typical cyclic disease, well known to every layman by the term "inflammation of the lungs." On account of the regular participation of the pleura it is also called *pleuropneumonia*, and on account of the nature of its appearance *primary, genuine pneumonia*. Less properly, the disease is designated as *lobar pneumonia* since bronchopneumonia also appears in a lobar form in addition to a number of other acute pulmonic inflammations having a

different etiology (partly streptococci and staphylococci), and which also often deviate in their clinical course by typhoid symptoms, by angina, prolonged course, termination by lysis, tendency to abscesses, and gangrene. It is justly desirable to separate these forms of pneumonia as *atypical pneumonias* from the croupous variety (Aufrecht), and in the future we will certainly also learn to differentiate them more readily clinically.

Etiology.—Croupous pneumonia is an infectious disease, a fact which has been made absolutely certain by the researches of Fränkel and Weichselbaum. Its cause is the *Fränkel-Weichselbaum diplococcus pneumoniæ* (often merely called pneumococcus), which in the majority of cases of typical croupous pneumonia is found in great numbers in the lungs, often in pure culture, frequently also with strepto-, staphylococci, Friedländer's and colon bacilli. The pneumococcus is also regularly found in the complicating diseases of croupous pneumonia, in pleurisy, pericarditis, peritonitis, meningitis, otitis, arthritis, and osteomyelitis. Moreover, all these affections may also be called forth apparently primarily by the pneumococcus. Often it may also be demonstrated by proper methods in the blood of patients suffering from pneumonia, (23 per cent. of cases, Schottmüller). In pneumonia, the pneumococcus is frequently found in the peritoneal cavity without peritoneal symptoms (Stooss). Besides the pneumococcus, but much more rarely, the Friedländer pneumonia bacillus may also produce the picture of a croupous pneumonia, perhaps also the streptococcus mucosus.

The Fränkel pneumococcus, a slender, oval, often lance-shaped, pointed coccus, appears mostly as a diplococcus, at times arranged in rows of 4–6, and is surrounded by a capsule (without it in the culture). It is found in large numbers in the affected portions of the lungs, as well as in the sputum of pneumonia patients; less numerous but very frequently in the nose, pharynx, and bronchi of healthy individuals. It stains readily with aniline colors, according to Gram. It grows only at the temperature of the blood in the culture (agar-agar, blood serum). For authentication, rabbits are inoculated, which rapidly die from sepsis.

While at the present time croupous pneumonia is generally regarded as a bacterial infectious disease, nevertheless it is certain that in the developing of the same, the *predisposing causes*, which formerly were solely held responsible, play a very important part. Duerck has shown by experiment on animals, that for the development of pneumonia, a definite traumatism is necessary; inhalation of dust, or more particularly a cold. In children we also not infrequently see a pneumonia develop in the course of a severe cold, under the influence of which the domiciled pneumococci may develop undisturbed. Duerck found pneumococci twelve times in thirteen healthy children. For the development of pneumonia, therefore, no new infection is at all required.



I. Ernst K., 7 months, died from pneumonia.
The entire lung, especially the lower lobes, infiltrated with small gray hepatized areas.

II. Helene N., 3½ months, died from pleuro-pneumonia.
In the lower lobes are sharply circumscribed hæmorrhagic areas. The pleura is covered with a yellowish-white exudate.

This relationship also explains without any trouble the development of pneumonia as the result of an injury (blow on the chest), which has been observed from time to time (also by myself), and in which the period of incubation is only of a few hours' duration.

The *seasons* of the year exert a marked influence on the frequency. Everywhere in Middle Europe the maximum number of cases is found in the spring, the minimum in the fall (see Fig. 71). Besides the injurious changes of temperature during the spring, the lasting impairment of the respiratory organs during the winter is principally responsible. During the spring of the year, there is an accumulation of cases, appearing almost epidemically as the result of grippe. Epidemic and endemic occurrence is also seen at other times. Jürgensen straightway designates pneumonia as a "house-disease."

Fig. 71.

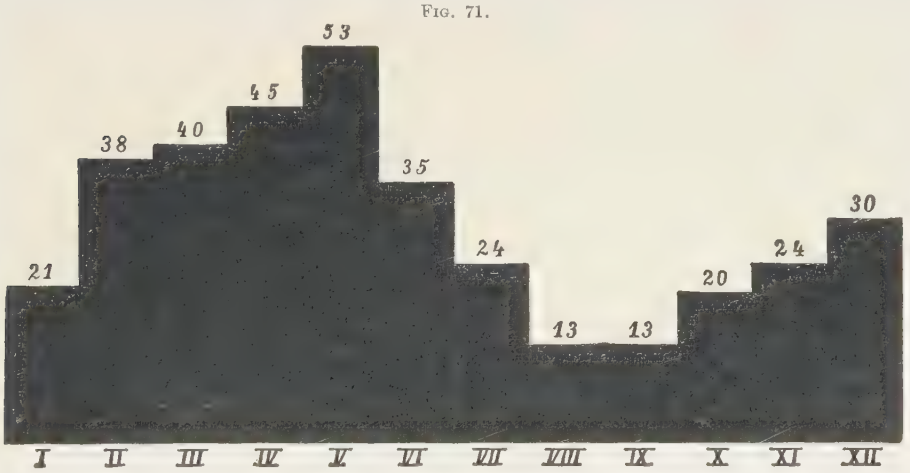


Diagram of frequency of croupous pneumonia according to months. From January to December (356 cases from the Children's Hospital of Paris, Comby).

Pneumonia very frequently occurs secondarily in grippe; less frequently in measles, typhoid, and other infectious diseases.

The period of childhood is directly subject to croupous pneumonia; more so than adult life. The formerly accepted view that younger children are not attacked by it is absolutely erroneous. Exceptionally, it has been observed congenitally (in pneumonia in the mother). Although the disease rarely occurs before the third or sixth month of life, it becomes quite frequent during the second year, and reaches its maximum from the second to the fifth year. Boys are more frequently attacked than girls, robust children just as well as debilitated ones. Although not very frequent, individual predisposition is undeniable, and may lead to three or four attacks in a child during the course of one or more years.

The **pathological anatomy** is exactly the same as in the adult and will, therefore, be but slightly dwelt upon. The disease generally affects

an entire lobe or at least the greater part of one. In the first stage (engorgement) and intense hyperæmia of the lung tissue is present. The alveolar epithelia are turbid and swollen, and a serous exudation begins to take place into the alveoli. In the second stage (red hepatization) the affected portion of the lung is large, bloodless, heavy, and tough (like liver). On section it is found to be dark red and coarsely granular. The prominent granules correspond to the contents of the alveoli and consist of a conglomeration of red and white blood corpuscles, detached alveolar epithelium, pneumococci in enormous numbers, the whole agglutinated by a network of fibrin. In children the granules measure only 0.07–0.11 mm. in diameter (Damaschino). In the bronchioles, fibrinous coagulum is frequently found. In the third stage (gray hepatization) the lung is still more expanded (costal impressions) and anæmic from compression of the vessels. The red blood corpuscles have disappeared and in their stead more and more white ones, rapidly undergoing fatty degeneration, appear. In the fourth stage (resolution) the lung becomes soft, more and more yellow, the exudate purulent in character, and the granulation diminishes. The contents of the alveoli disappear by absorption, less by expectoration, and again become filled with air. The pleura is almost always also involved in the shape of a fibrinous, and later often a serous or purulent, pleuritis.

General Clinical Picture.—In older children, from about the seventh or eighth year on, the beginning of a croupous pneumonia is, as in adults, *sudden*, with chill, and pain in the side. In younger children, to whom the following description has particular reference, pneumonia sets in suddenly, though a chill seldom occurs; but in its stead, slight chilliness and pallor. Very frequently the disease commences with violent vomiting, sometimes but more rarely, with convulsions. On careful inquiry, symptoms generally accepted as prodromal are mentioned. Lassitude, discomfort, and a slight cough, have preceded the onset for several days. If pneumonia occurs during an attack of grippe, it usually develops only after a febrile bronchitis of several days' duration. Whenever the disease occurs secondarily in the course of measles and diphtheria, the onset is often not very striking.

With the beginning of a pneumonia, the patients become very ill and request to be put to bed. A high fever manifests itself by sparkling eyes, reddened cheeks, burning skin, and a tense and very frequent pulse. Respiration is accelerated, but is not, or only slightly, dyspnœic; expiration is often somewhat interrupted and moaning, from the very beginning, but not always extraordinarily so. Immediately after the beginning of the illness, older children complain of pain in the side. Those of from three to seven years of age mostly refer the pain to the upper abdominal region; still younger children do not manifest any kind of local pain, or cry when picked up. The cough is slight or absent in

the beginning, there is no expectoration, and physical signs are absent in the lungs.

The symptoms of a pneumonia in young children during the first and second day, therefore, are apt to lead the inexperienced one to think rather of some general febrile disease. In the meanwhile the temperature rises to 39° C. (102° F.) and 40.5° C. (105° F.) with restlessness, sleeplessness, thirst, and loss of appetite. In pneumonia involving the upper lobe, the first signs of consolidation (a tympanitic note, dulness, bronchophony and bronchial breathing) are found on careful examination in the region of a lobe, very often only in the fourth or fifth day or even later. These lead to a proper diagnosis.

Frequently also, only the appearance of a herpes labialis (see Fig. 72) on the third or fourth day induces an examination of the lungs. About this time, too, the external symptoms which point to a croupous pneumonia become more apparent. The respiration is markedly accelerated, with dyspnœa and dilatation of the nostrils. Moderate inspiratory recessions occur, particularly in younger children. Expiration is noticeably suppressed and is of a well-marked grunting and moaning character. The grunting expiration is not so plain during rest as while speaking and crying, and sound interrupted, suppressed, and painful. Continuous crying is avoided. If cough be present it is short and suppressed, and is often followed by crying in consequence of the pain which is produced.

Fig. 72.

Herpes of the mouth and lips in croupous pneumonia. Boy six and one-half years old.

The clinical picture changes somewhat towards the end of the week. The high, continuous fever, together with the lessened amount of nourishment which has been taken, have already preceptibly thinned the cheeks of the patient. The patient loses in strength and no longer attempts to sit up alone. The facial expression is anxious and painfully distorted. The respiration has become somewhat more accelerated and is more laborious. The pulse becomes more frequent and softer. The tongue is heavily coated and dry, bowels are usually sluggish, and the urine is scanty and dark in color. The appetite is still more diminished. The physical signs in the lungs have become very plain by this time. From the fifth to the sixth day on, apathy, somnolence, occasionally also delirium and alarming weakness, often occur. Then, mostly at this time the longed-for crisis occurs which is frequently announced by the

appearance of crepitant râles and by the skin becoming moist. The temperature which during the whole course of the disease has been continuously high, ranging from 39° and 40° to 40.5° C. (102°, 104°, 105° F.) drops to normal or somewhat below normal within 12–24 hours. This decline in temperature occurs between the fifth and ninth day, mostly in the seventh day. Occasionally, during the crisis the patient perspires very freely, and lapses into a quiet slumber. On awakening, the clinical picture is very much altered. Many children are then very bright, and ask for nourishment; others, who have been more ill are still very feeble and languid; but respiration has become much easier, slower, and noiseless, no longer grunting. The frequency of the pulse also diminishes rapidly. Dulness, bronchophony, and bronchial breathing, rapidly subside, and at the end of a week have usually entirely disappeared, leaving the patient fully convalescent.

Whenever the disease does not terminate favorably, and does not come to a crisis, perhaps because another lobe has become involved, the condition becomes constantly more serious from the eighth and ninth day on. The respiration becomes more labored, marked inspiratory recessions and cyanosis appear. The pulse becomes very small and intermittent, increasing to 200 and more. With increasing stupor and weakness or pulmonary œdema, death occurs, usually during the second week.

Even after resolution has taken place dangers may still threaten. In the first place from pleurisy (often purulent) or pericarditis. In rare cases, also, pneumonia terminates in atrophy. Termination in gangrene of the lungs which occurs exceedingly seldom is scarcely ever dependent upon a pneumonia *per se*, and may be more properly attributed to inspiration of particles of food. The same may be said of abscess of the lung which is rarely observed, and which, may, however, also develop from an encapsulated, purulent, pleuritic exudate.

Individual Symptoms and Complications.—From the beginning, *respiration* shows a marked increase in frequency. During the early years of life this often attains 50–60, yes, even 80; in older children 40–60–70. Since in the beginning, signs of dyspnœa are frequently absent, one is easily disposed to blame the fever solely on the increased frequency of respiration. On careful examination, however, the respiration proves to be more rapid than the pulse, so that the ratio is less than 1 : 3. On expiration a moderate retardation or dragging of the affected side is shown. This, however, is never as marked as in exudative pleuritis, and is particularly absent in the beginning. In cases of involvement of a lower lobe, mensuration may show a slight expansion of the corresponding half of the chest (Ziemssen).

The grunting, moaning and noisy expiration, is a particularly frequent symptom in the subsequent course of croupous pneumonia. It is seldom found so outspoken in other diseases, or in bronchopneumonia,

so that its appearance should at once arouse a suspicion of croupous pneumonia. Experienced mothers frequently make a proper diagnosis from this symptom alone. It is probable that the grunting expiration depends chiefly upon an involvement of the pleura. This explains why in a central pneumonia (to which many apex pneumonias belong especially) it is so frequently absent. After the crisis the respiration rapidly becomes quiet and regular, dyspnœa disappears, and the number of respirations becomes normal within a few days. In the beginning, dyspnœa is often scarcely noticeable, and even later, especially in central and apical pneumonias, it is but slightly marked. As a general rule, it is less marked than in bronchopneumonia and for this reason, cyanosis and inspiratory recessions are of a lesser degree. In extensive consolidation and particularly in an attendant bronchitis the dyspnœa may assume a very high grade.

Pains in the chest are on an average less than in adults and are frequently absent, especially in involvement of the upper lobe and in central pneumonia. Older children complain of them next to the sternum when the middle lobe is affected. During the first two years the symptom of local pain is frequently absent, and sometimes percussion is unpleasant. Children of from three to seven years of age very often refer the pain to the epigastric region. These gastric and abdominal pains are decidedly characteristic of croupous pneumonia. Not infrequently, these pains simulate an appendicitis, to which also the vomiting, the absence of physical signs in the lungs, and the appearance of grave clinical manifestations in the midst of perfect health, are added in a misleading manner. They have led to operation in these cases of "*pneumonic pseudo-appendicitis.*" The abdomen, however, is not sensitive to pressure.

The *cough* is often slight or may be entirely absent in central and cerebral pneumonia. It is painful, and for this reason, short, suppressed, and is frequently followed by crying. The cough is most marked and frequent during and after defervescence. The sputum is absent before the eighth or the eleventh year. Sometimes, vomiting produces the typical rusty sputum.

From what has been stated, the general symptoms in croupous pneumonia in younger children are frequently less prominent, and the three cardinal symptoms of this disease in adults, chill, pain in the side, and red sputum, may be absent. It is, therefore, of greater importance in children, to make *repeated, careful, daily, physical examinations.* In this connection several important points in general must be mentioned which frequently do not receive sufficient attention.

Auscultation is the most important part of the examination, and often renders a diagnosis possible earlier than by percussion. On account of the pleuritic pains, patients breathe superficially, so that

vesicular breathing and râles are frequently marked and become prominent only on forced respiration. It is, therefore, of unusual advantage to examine the lungs of an infant during crying or coughing. Ziemssen (1862) has emphatically pointed out that the auscultation of the voice in children is very important, and that bronchophony is a much more valuable sign than bronchial breathing and tympanitic râles. In painful and, for this reason, superficial breathing the sounds in young children often cannot be determined; while on the other hand during crying and coughing the voice is well transmitted to the periphery of the lungs, provided there is no occlusion of the bronchi. For this reason we can examine for bronchophony much easier and earlier than for bronchial breathing.

Now, as far as an auscultation in croupous pneumonia in particular is concerned we find that crepitant râles are only seldom heard in the beginning, but on the other hand, very frequently during the stage of resolution at the time of the crisis. Over the affected lobe the puerile respiration is at first diminished or increased, and undetermined. If the inflammation affects a lower lobe, *bronchophony* and *bronchial breathing* are heard primarily on the second or third day, more rarely as early as the first day, or after the fourth day, usually along the vertebral column in the region of the root of the lung. In pneumonia of the upper lobe, bronchophony and bronchial breathing are generally heard posteriorly over the spine of the scapula, and may be absent until the fourth or sixth day, in certain cases even until after the crisis. Frequently also, bronchial breathing is very distinct during inspiration. In central pneumonia dulness is often absent and bronchophony is the only certain sign of consolidation. Otherwise, the extent of a pneumonia may be more accurately mapped out by bronchophony than by percussion (Ziemssen).

In the region of the upper dorsal vertebræ and to the right thereof, bronchophony and bronchial breathing are heard normally to a varying degree, and still more so when enlarged bronchial glands are present. Experience and daily comparative auscultation where there is a suspicion of pneumonia protects against error under these circumstances. In very extensive pneumonia of the one side, one occasionally also hears bronchophony and bronchial breathing on the sound side. One must never neglect to examine also the sides of the thorax, as in the region of the axillæ, in particular, many pneumonias first manifest themselves. After the crisis, bronchial breathing and bronchophony usually disappear in the course of a week.

In percussing, we must percuss lightly and strongly alternately, in order to recognize both superficial and deep seated areas. In central pneumonia so frequent during childhood (pneumonia very often begins or remains central especially in the upper lobes) percussion may remain negative or uncertain during the whole course of the disease. Usually,

however, tympanitic, and soon diminished tympanitic, resonance appears over the area of one or more lobes of the lung. Indeed, like the auscultatory phenomena, it appears, as a rule, earlier in pneumonia affecting a lower lobe, and is then more distinct at first towards the root of the lung. It appears later in pneumonia of the upper lobe where it is first heard distinctly over the spine. During the further course of the disease, dulness becomes more and more marked, more resisting, and no longer sounds tympanitic. It again becomes tympanitic only during resolution, and usually disappears completely during the first week after the crisis. The dulness often corresponds to the border of a lobe, the anterior portion of which may, however, remain free. The resistance is not so marked as in pleuritic effusion. A tympanitic percussion note is frequently found normally over the lungs of children, often transmitted from the stomach (in this instance it may also be metallic), or from a gut distended with gas. It has not as great significance as in the adult. The tympanitic resonance is often very distinct in front below the clavicle in pneumonia of the posterior portion of the upper lobe or of the lower lobe. The vocal fremitus is often increased over the consolidated lung, provided the bronchi are not occluded. It is obtained by placing the inner edge of the hand on the chest, or by auscultation. The examination is rendered difficult from the fact that under normal conditions fremitus is not distinct with ordinary voice in children under six to eight years of age, at least not posteriorly where it is of the most importance. During severe crying or coughing, on the other hand, fremitus is obtained even in infants, and, indeed, is often increased over a consolidation.

The *pleura* participates regularly in croupous pneumonia as soon as the inflammation reaches it.* Friction is less frequently heard than in adults, very likely because the movements of the thorax are curtailed by the pain, a condition which the physician cannot increase at will. The most frequent and most important complication is exudative, and especially purulent, pleuritis, during which bronchial breathing and bronchophony may be very distinct and not only continue to be present but may even be increased.

The *pulse* is always very much increased in rapidity, and during the first years of life and during high temperature may reach 180–200 without causing an unfavorable prognosis. It is more unfavorable when the pulse becomes too small, intermittent, and easily compressible at the time of the crisis. After the crisis, the frequency diminishes rapidly, but often only becomes normal after several days. An irregular and slow pulse often occurs in older children during convalescence and is not a bad symptom.

The *heart* of the infant on account of its still unenfeebled condition,

* Schlesinger found in 173 cases of pneumonia, pleuritic symptoms in 51 cases, distinct effusion in 16, of which 7 were purulent.

generally guarantees a favorable termination, the strong right ventricle contributing especially thereto. A fatal collapse during the period of convalescence is an unusual event. A not very infrequent and a very serious complication is the occurrence of *pericarditis*, often fibrinous, purulent and causing a fatal termination. During life, pericarditis, which often develops with pleurisy, is frequently unrecognized (von Jaksch). Severe cyanosis, dyspnœa, a bad pulse without sufficient reasons, according to the condition of the lungs should lead one to think of this complication. Endocarditis is less frequent and not so serious.

From the first day of the disease, the blood shows a marked *leucocytosis* (instead of 10,000 leucocytes as high as 50,000 to mm.^{3}), which increases until the crisis is reached and then rapidly decreases (v. Jaksch, Monti, Berggrün).

Leucocytosis is of prognostic value, since in fatal cases it is often relatively low or absent. Polynuclear cells predominate.

The *digestive organs* show nothing special except the frequent initial vomiting. The tongue is heavily coated, the appetite is poor, the thirst is great. Older children are frequently constipated; in infants diarrhœa occurs at times. A follicular tonsillitis sometimes precedes a pneumonia. The liver, and more frequently the spleen, may become enlarged to a moderate degree. Exceptionally a purulent peritonitis may develop, which like most of the complications is caused by pneumococci, the prognosis being comparatively favorable. Jaundice is less frequent than in adults.

The *urine* is scanty and high colored. It is deficient in sodium chloride, rich in urates, and also rich in urea which may still increase after the crisis owing to the resorption of the pulmonary exudate. A febrile albuminuria is frequently present, less often a genuine acute nephritis which is at times hæmorrhagic in character. Peptone, acetone, and acetic acid, are frequently present in the urine. Diazo reaction is not frequent.

The *nervous system* frequently shows a serious participation of the brain which often dominates the entire clinical picture (cerebral pneumonia, etc.). In older children, delirium is often observed near the crisis, and postpneumonic melancholia. Aphasia and unilateral palsies have also been described (Aufrecht). Genuine purulent meningitis is a rare complication. Pfaundler frequently found the patellar reflex diminished or absent, often even before the occurrence of the pulmonary symptoms. He regards this symptom of diagnostic value.

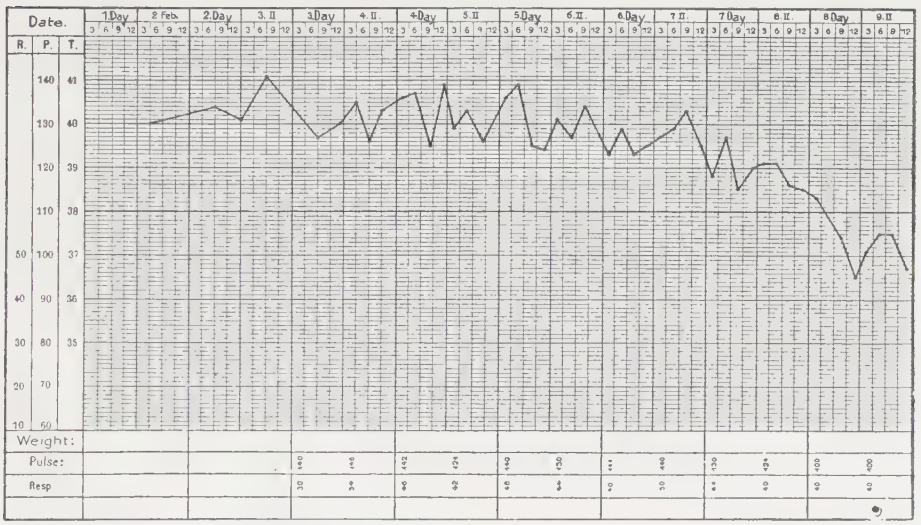
The Skin.—Herpes facialis is decidedly more rarely found than in adults (see Fig. 72). The statements regarding the frequency of the same vary considerably. Schlesinger found it in 18 per cent., Comby in 10 per cent. of cases. Especially during the first years of life, when it would be desirable for diagnostic purposes, it is frequently absent. It may appear after the third or fourth day. An intense circumscribed

redness of the cheek is often seen with the onset of the fever, and frequently only disappears after the completion of the crisis. The redness of the cheek is frequently unilateral, but does not by any means always correspond to the seat of the pneumonia. In some cases, at the commencement of the disease, a general erythema of the skin mostly scarlatinal in appearance occurs, but is of no consequence (Macé).

The sweating which usually takes place at the time of the crisis is usually less severe than in adults, though sometimes causing severe sudamina of the trunk, often followed by mild desquamation.

Otitis media is a rather frequent complication, and often assists in the presentation of the picture of a cerebral pneumonia. It readily

FIG. 73.



Croupous pneumonia of the left upper and lower lobes and a portion of the right lower lobe. Seven-year-old girl (typical temperature curve, rectal temperature).

leads to suppuration and perforation of the tympanic membrane. During a grippe epidemic it is very apt to complicate croupous pneumonia.

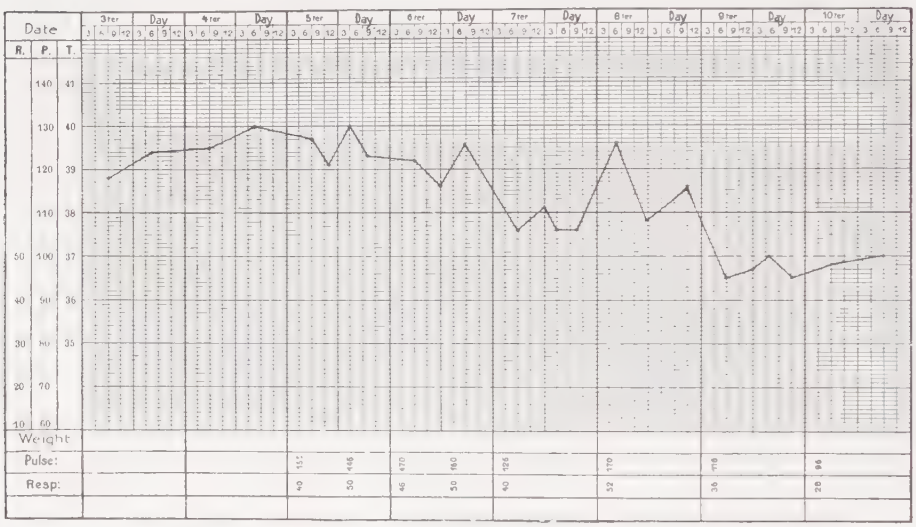
Purulent osteomyelitis and *arthritis* (Hagenbach-Burckhardt and their pupils Meyer and Pfisterer) develop not very infrequently as true metastases in connection with croupous pneumonia, usually during the first two weeks, and are more apt to affect young infants, frequently in addition to a purulent pleuritis. Most frequently, the larger joints (shoulder, knee) are affected. Abscesses in the skin are frequent. The course of these affections, which contain the pneumococcus in pure culture, is relatively benign and recovery often takes place after simple incision. Analogous purulent metastases also often develop from a primary pneumococcus otitis.

Temperature Curve and Duration.—In typical cases of croupous pneumonia in children, the temperature is high and continuous from

the beginning. It ranges from 39.5–40° C. (103°–104° F.), increases towards the end of the week, and, between the fifth and ninth day of the disease (see Fig. 73), drops within 12–24 hours to normal or somewhat below (crisis). Ziemssen found the beginning of the crisis to occur mostly in the second half of the seventh day, or occasionally on the fifth day, less often on the third, ninth, eleventh day, seldom on days of even numbers.

A remission often occurs on the fifth day, less often on the third. With relative frequency, a rapid decline of the temperature to as low as normal may occur the day preceding the crisis (pseudo-crisis). This Baginsky has designated as pro-critical decline (see Fig. 74). Following the crisis the temperature frequently remains subnormal for several days, and may show one or two evening exacerbations.

Fig. 74.

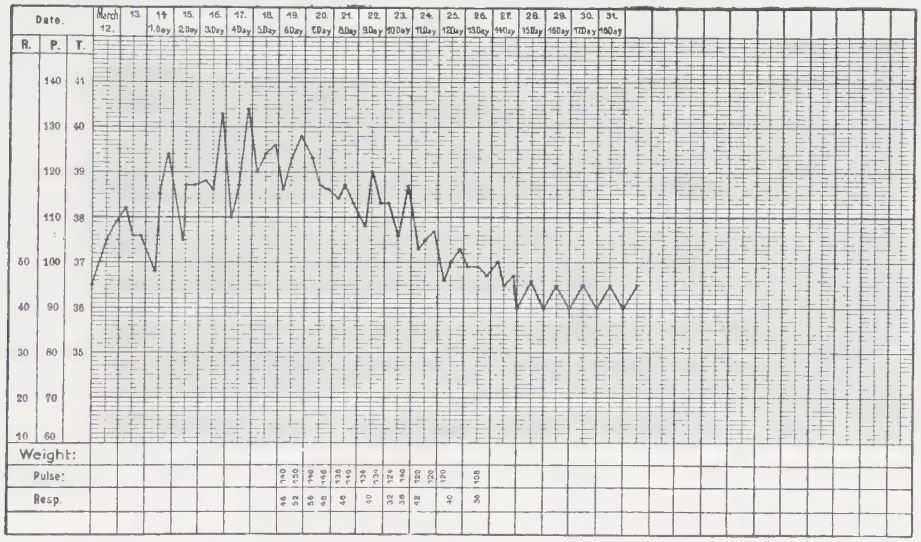


Croupous pneumonia of the left lower lobe with pseudo-crisis. Boy, two and a half years old.

From this typical temperature curve there are many departures. The duration of the fever may in exceptional cases last only one or two days. At times it may extend over a period of 12–14 days, or even as long as three weeks, in which case either an upper lobe is usually involved, or it is due to an involvement of another lobe. Pneumonia affecting the upper lobe is frequently characterized by an excessively high temperature. In younger children the course of the temperature is often comparatively remittent or intermittent, most frequently in pneumonia of the lower lobe. Not so very infrequently, a seeming crisis is followed by a moderate or high elevation of temperature lasting several days, after which the definite crisis takes place. In a number of cases the fall of the temperature occurs by lysis (12 per cent. Schlesinger). But the decline by lysis is often suspicious of pleuritis as a complication.

Seat of the Pneumonia and Peculiarities of Course.—Croupous pneumonia often affects a lobe in its entire extent. Frequently, however, it involves only a portion of the same, and permits other parts (especially the anterior) to remain free. At times it extends to one or two lobes of one or both sides. More frequently than in adults, pneumonia is limited to a single central area. According to three extensive statistics (Rilliet and Barthez, Baginsky, Comby) each embracing more than 300 cases, there is a remarkable coincidence in regard to the localization and participation of the individual lobes. The upper lobes are affected as frequently as the lower. Most frequently affected are the right upper lobe and the left lower lobe,—the right upper lobe at least twice as often as the right lower lobe. Quite frequently also the middle lobe is affected;

Fig. 75.



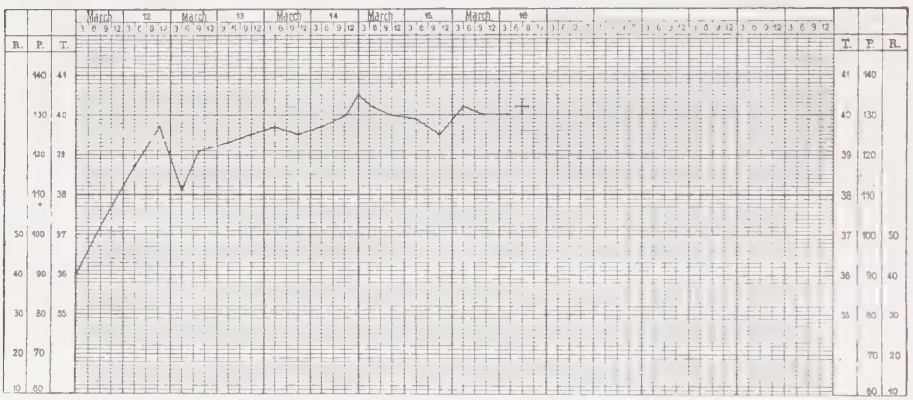
Migratory pneumonia in grippe (right upper and left lower lobes). Female (twin), twenty-two months old.

not uncommonly both lungs are involved. During the first years of life, the upper lobes are more often attacked. Before the crisis takes place in the one lobe, the inflammation at times extends to another lobe, and from here sometimes to a third lobe by which the disease may be prolonged by fourteen days or even longer. Such migratory pneumonias are not uncommonly observed in grippe. One readily falls into the error of assuming that a migratory pneumonia is present whenever a pneumonia attacks two lobes simultaneously, which however is not apparent in both situations at the same time. Croupous pneumonia complicating grippe, often several days after the disappearance of fever, coryza, and bronchitis, has the peculiarity that it is very apt to appear as an apical pneumonia, producing a varying temperature which usually drops by lysis to the normal in two or three days (see Figs. 75–76).

Relapses in pneumonia are of rare occurrence. A fresh pneumonia sets in one or more days after the crisis has taken place.

An *abortive type of pneumonia* is spoken of whenever the disease sets in like an ordinary pneumonia, but in which the temperature begins to drop on the fifth day, an event which occurs more frequently in children than in adults. A duration of only two or three days is not at all uncommonly observed. Even cases of only one or two days' duration have been definitely established, debarring cases in which death occurred as early as the first day (v. Dusch). I, myself, have seen a pneumonia lasting only one day in a seven-year-old girl, in which the diagnosis was confirmed by the rusty colored sputum brought to light by vomiting. In cases where the crisis takes place after only a few days, the signs of consolidation often appear only later, and may last for days. In contradistinction thereto are the remarkable cases observed by

Fig. 76.



Croupous pneumonia in grippe. Girl, twenty-two months old, becoming ill simultaneously with her twin sister, see Fig. 75.

Henoch in which the physical signs in the lungs had already disappeared before the crisis. The abortive pneumonias permit the conclusion that the process of inflammation may come to a standstill at any stage of the disease, even at the stage of engorgement. The cases of pulmonary congestion described by Cadet de Gassicourt are probably abortive forms of pneumonia.

A type of pneumonia frequently seen in children, and described by Rilliet and Barthez as *cerebral pneumonia*, is characterized by marked cerebral symptoms at the beginning or during the subsequent course of the disease. An eclamptic and a meningeal form are distinguished. The eclamptic form is chiefly seen in children during the first two years of life. General convulsive seizures occur not only as initial symptoms, but are also common during the subsequent course of the disease. Spasm of the muscles at the back of the neck, apathy, and somnolence, are usual accompaniments. The meningeal form is more apt to appear in

children from three to seven years with headache, violent vomiting, stupor and coma, rigidity of the neck, delirium, and hyperæsthesia of the skin. The pulse is not slowed, nor are the pupils dilated. Such cases are more apt to mislead one into accepting the presence of a genuine cerebral affection, for the reason that these symptoms are produced by a central pneumonia, the evidences of which become manifest only towards the crisis. According to Schlesinger, cerebral pneumonia is seen just as frequently in pneumonia of the lower lobe as in apical pneumonia; while according to the statements of most authors it is found mostly in apical pneumonia. As a rule, the prognosis is good, but the course is apt to be severe. Otitis media is relatively frequent in these cases, and probably is partly also responsible for the cerebral manifestations. Furthermore, a *gastric* and a *typhoid* pneumonia have been distinguished. But the distinction is of little value, and simply goes to show that the pulmonic symptoms in children frequently step into the background and are followed by those from the stomach or general symptoms, in this way dominating the clinical picture.

In this connection, *splenic pneumonia*, also called *massive pneumonia*, may be mentioned for diagnostic reasons. It was first described by Grancher in 1883. Until the present time it has received consideration only in French literature. I, myself, am lacking in personal experience regarding this peculiar disease, which is characterized by the fact that it presents a form of acute inflammation of the lungs, but bears the marks of an exudative pleurisy. According to Queyrat the disease occurs not uncommonly in children, mostly in boys, chiefly during tuberculosis and grippe, and usually follows a cold. High temperature, moderate dyspnœa, and violent cough without expectoration even in older children, rapidly set in. Absolute flatness is found mostly at the lower portion of the left lung, with disappearance of vocal fremitus, marked bronchial breathing, and ægophony, symptoms which, therefore, point to pleurisy with effusion. In favor of this view also is the frequent disappearance of the apex beat. But symptoms against pleurisy are fine crepitant râles, exemption of Traube's space, and absence of displacement of the sternum to the affected side. The pulmonic symptoms disappear very gradually, only after 8 or 10 or even after 15 days. The disease almost always terminates in recovery. As long as no definite etiological and anatomical findings are submitted in this remarkable disease it must be accepted with a certain amount of skepticism.

The **diagnosis** of pneumonia is usually easily made in older children, since nearly all symptoms with the exception of the sputum are typical, —the same as in adults, viz: diminished and bronchial breathing, bronchophony, dulness, and increased vocal fremitus. On the other hand, the diagnosis in young infants presents many difficulties where chill, cough, and pain in the side are absent, herpes labialis occurring less

frequently than in adults. Instead of these, abdominal pains and symptoms from other organs may predominate. It may readily be understood that beginners usually do not think of pneumonia at all, and rather assume anything else (gastric fever, meningitis, etc.). This is quite pardonable, since the objective symptoms often are not present until from the third to the sixth day. This is particularly the case in apical pneumonia, which also in many other respects presents the most difficulties for diagnosis, as is sufficiently shown by the above description of the disease.

In bronchopneumonia the question usually hinges on whether only a bronchitis is present, or in addition thereto a pulmonary consolidation; in other words, whether a bronchopneumonia is present or not. In croupous pneumonia the difficulty is mostly as to whether a pulmonary affection is present or not.

In cases where there is a sudden elevation and a continuous temperature the possibility of a croupous pneumonia must be constantly born in mind, even when no symptoms point directly to the respiratory apparatus. Indeed, pain and cough are frequently absent, and one is prone to attribute the accelerated respiration to the temperature. Usually, however, the *characteristic grunting expiration* is present, and also a somewhat dyspnœic (labored) respiration which directs our attention to the lungs. One must make it a rule to make daily examinations of the lungs in all cases of high temperature. By doing so, unimportant symptoms, which were not previously present, will be promptly and correctly interpreted, thus avoiding delay in diagnosis of a croupous pneumonia, a suspicion of which is only sometimes aroused by the sudden decline of the temperature on the fifth to the seventh day, when it is usually easily established. Whoever does not make it a rule to search for croupous pneumonia (especially also over the scapulæ), will at times overlook this disease, and perhaps only later will be able to properly estimate the previous condition by the occurrence of a purulent pleurisy; furthermore, systematic taking of the temperature is valuable for diagnosis, inasmuch as a high and continuous temperature is seldom seen under any other circumstances.

Bronchial breathing and *bronchophony* over a circumscribed area, where they were previously absent and which disappear again after a few days, are frequently the only pulmonary physical signs in central (especially in apical) pneumonia, and are sufficient to establish the diagnosis which often can be made with considerable certainty many days previous to the late manifestations of pulmonary consolidation, namely, by the sudden onset with high continuous temperature, and by the accelerated and somewhat dyspnœic breathing with grunting expiration. The absence of bronchophony and bronchial breathing in pneumonia is frequently due only to the fact that the infant breathes super-

ficially. Crying and coughing with the resulting deep inspirations will then cause these symptoms to become plainly manifest.

One should always remember that in percussing a crying infant, and especially if it is pressing down, that a slight dulness may be present over the lower and posterior portions of the lungs under normal conditions. But this disappears again on inspiration, and therefore may be easily distinguished from a dulness resulting from a pathological condition. Furthermore, there is frequently found at the pulmonohepatic border behind and on the right side greater dulness than in the left. This is caused by the large size of the liver. Those who are inexperienced are apt to regard this relationship as dulness within the thoracic cavity. The normal vesicular breathing readily shows, however, that no pathological condition is present, and confirms the view that this dulness has its seat within the range of the liver. In small children the spinous processes are counted with difficulty. It is more practical under these circumstances to determine the lower border of the lungs behind, according to the ribs. It is normally found at the tenth rib in the posterior scapular line, and is easily determined by counting from the twelfth rib.

Owing to insufficient pulmonic findings, a *differential diagnosis* must be made in many febrile infectious diseases in their beginning. When cerebral symptoms are present one readily thinks of meningitis. But *tuberculous meningitis*, which on account of its great frequency is likely to be thought of first, develops slowly; and, moreover, convulsions and stupor are not present in the beginning as in cerebral pneumonia, but come on later in the course of the disease. The temperature in tuberculous meningitis is mostly moderate and varying in character. The meningeal symptoms in croupous pneumonia are incomplete; irregular and retarded pulse, dilatation of the pupils, and paralysis, are almost always absent.

Typhoid fever in children may begin with and have throughout its course a high temperature; so that for days it will be impossible to arrive at a definite conclusion until, on the one side, pulmonary symptoms, and, on the other side, enlargement of the spleen, roseola, diazo- and Widal-reaction, become prominent. Until bronchial signs decide in their favor, grippe and influenza with high fever may for days raise a suspicion of croupous pneumonia, if the catarrhal conditions of the upper air-passages and the conjunctiva are not pronounced, and if a somewhat dyspnœic breathing as the result of swelling of the bronchial mucous membrane be present. Likewise, a simple bronchitis with high fever does not permit a differentiation from pneumonia during the first few days.

Whenever positive physical signs are found in the lungs, a differential diagnosis of bronchopneumonia, pleuritis, and acute tuberculosis of the lungs, is to be made.

The differentiation from bronchopneumonia, which has already been mentioned on page 347, can usually be made; although since the local symptoms are not always sufficient for this purpose, the development, course, and temperature curve must be considered. Still, there are cases—especially in measles and grippe—in which these two types cannot always be separated clinically. Even the anatomist is sometimes in doubt how he should classify a pneumonia which he has found to be present. In exceptional cases, bronchopneumonia and croupous pneumonia may co-exist.

We are frequently confronted at the bedside by the question whether a *croupous pneumonia or a pleurisy with effusion* is present, when dulness, bronchial breathing without râles, or with scanty fine râles, appear in the region of the lower and posterior portions of a lung; in connection with which the local physical signs, course, and temperature, are to be taken into consideration. In croupous pneumonia, dulness develops rapidly and almost simultaneously in its entire extent, and is at times more marked above than below. Almost always Traube's space remains free. In addition, the dulness disappears in a few days after the decline in the temperature. The sensation of resistance is not marked. In pleurisy, the dulness is greater and the sensation of resistance is more marked than in pneumonia. The dulness is most marked behind and below, and gradually rises above and to the side. If it is present behind about the middle of the scapula, dulness is also found anteriorly. In large effusions the neighboring organs (heart and liver) are displaced. If the dulness involves the entire side behind and in front we are dealing with a pleurisy in the majority of cases. The dulness gradually diminishes from above downwards. In croupous pneumonia loud bronchial breathing is most plainly heard where the dulness is most marked. Bronchophony is very marked. In pleurisy, bronchial breathing and bronchophony are less pronounced and softer, and are most marked at the upper border of the dulness. On the other hand, bronchial breathing is heard almost everywhere in extensive and recent effusions. In these cases the respiratory murmur is not abolished as in adults. Ægophony points to pleurisy. Increased vocal fremitus points directly to pneumonia; whereas, on the other hand, the fremitus often cannot be determined and is not infrequently diminished as a result of occlusion of the bronchi. In pneumonia, the respiratory movements of the affected side are only slightly limited; whereas, in pleurisy they are very markedly so, and in cases of extensive pleurisy the affected side is noticeably expanded even to the eye. Croupous pneumonia is characterized by a high, and after about 5–9 days, critical decline of temperature. Pleurisy, if it is not purulent in character, rarely causes a high temperature, at least, perhaps, only during the first few days. It then soon shows marked remissions, gradually becoming normal in from two

to four weeks. A pleuritic effusion, developing during the course of a pneumonia of an upper lobe and accumulating below, may readily simulate a fresh pneumonia.

Pleurisy with effusion may frequently occur in combination, or as a sequela of croupous pneumonia. In these cases, the temperature rises during the last days of a pneumonia, is prolonged or shows an imperfect decline by lysis, and in cases of subsequent increase often points to a purulent character of the effusion. The pulmonary dulness is increased by the simultaneous development of an effusion, the resistance is increased, and the fremitus is diminished or abolished. On the other hand, bronchial breathing and bronchophony are usually increased and not diminished as one would readily suppose. Displacements of neighboring organs occur.

Whenever the previous course is unknown and the consolidation rapidly increases, an acute tuberculous pneumonia may readily pose as a croupous pneumonia. In these instances, the absence of sputum and the fact that tuberculosis in young infants frequently begins in a lower lobe renders the diagnosis more difficult. A rapid course, with severe dyspnœa and cyanosis without adequate physical signs in the lungs, often points to miliary tuberculosis. The previous condition of the lungs, course, and temperature, determine the diagnosis in these cases.

Hæmorrhagic infarct is not frequent in children (in mitral lesions). The symptoms are similar to a circumscribed, croupous pneumonia; but in embolism the fever is either absent or moderate. Owing to the absence of sputum the diagnosis is rendered difficult.

Chronic pneumonia and *chronic pleurisy* may sometimes be mistaken for croupous pneumonia, whenever, without a complete history, they come under treatment simultaneously with a febrile affection. The subsequent course of the disease will clear up the diagnosis; but, beforehand, the retraction of the affected side of the thorax should lead to the proper recognition of the existing condition.

Prognosis.—Of all severe diseases of childhood the prognosis in croupous pneumonia is probably the most favorable. The mortality only amounts to a small percentage (3–5) and chiefly falls on the first, less on the second year of life. The prognosis is, therefore, very much better than in bronchopneumonia, and the treatment more satisfactory to the physician. Vigorous and previously healthy children very rarely succumb to croupous pneumonia. A delicate constitution, rachitis, and preceding acute infectious diseases (measles, typhoid fever, whooping-cough), may prove dangerous. The most important complication, purulent pleurisy, is usually cured by early and proper treatment. *Pericarditis* is a frequent cause of death. A very high temperature with severe dyspnœa, crisis delayed beyond the 9th–11th day, and an unusually protracted course of the disease, make the prognosis as to recovery

gloomy. In individual cases, a general sepsis from pneumococcus infection causes death (Vierordt).

Prophylaxis is of less value than in bronchopneumonia. Nevertheless, even in these cases, a general, rational, hygiene and hardening are certainly of value, as are the protection of the organs of respiration from dust, and also the care of the mouth. Not infrequently, a mild catarrh precedes a pneumonia. Severe colds are to be avoided; for instance, the rapid cooling of the perspiring body. It is wise to separate healthy individuals from one who is suffering from pneumonia, although positive evidence of direct contagion has never been submitted. Epidemics may also be explained on other grounds, and often occur in connection with atypical, non-croupous pneumonias. I have at times observed two members of a family become ill with pneumonia simultaneously, but always as the result of grippe. The tendency of croupous pneumonia to always recur in certain dwellings (pneumonia houses) is unexplained. Perhaps it depends upon the demonstrated longevity of the pneumococcus in its dried state. Accordingly, disinfection as a prophylatic measure would be in place in these instances. In children, there is seldom an opportunity for disinfection of the sputum.

Treatment.—The majority of cases of croupous pneumonia in children previously healthy recover under any treatment, in so far that it is not directly injurious. Copious blood-lettings, severe irritations of the skin, and powerful drugs, belong to the past. In robust children an expectant plan of treatment with mild hydrotherapy is amply sufficient. Nevertheless, we must exercise the greatest care in our treatment of every case, for we do not know whether the greatest demands will not be made upon the system by the duration of the disease or by complications.

Provision must be made for proper bedding, airy room, and an equable temperature. Rendering the air moist is necessary only during the season of the year when the rooms are artificially heated, and never to such a degree as in cases of bronchopneumonia. Besides the care of the mouth and skin, attention must be paid to the diet. The diet should be free, but only fluid, and for infants diluted. Older children may be allowed eggs, tapioca, farina, softened rusk, and fresh fruit juices from the beginning. Whenever it is indicated, the nutrition may be increased by the expressed juice of meat. An abundant supply of water is of importance even in the form of sugar water and infusion of lime-blossoms (if necessary by rectal irrigation), especially in cases with typhoid and cerebral symptoms, for washing out the bacterial poisons. In older children the quantity of urine voided daily is an important criterion for the quantity of water to be given, and for the cardiac strength.

Lukewarm *baths* are the most agreeable remedy for temperature, and the local and general symptoms of the disease. Whenever the tem-

perature exceeds 39° C. (102° F.) a bath two or three times daily is given; in cases of infants at 27° R. (93° F.); in older children as low as 25° R. (89° F.). Duration 6–15 minutes. In addition, cold sponging of the entire body followed by dry friction is often advantageous. With this treatment, one can get along in mild cases. Besides the baths, or in the first instance *cold chest compresses* are very popular, and are of decided value in prolonged temperature over 39° C. (102° F.). Regarding cold and warm compresses and baths and their contraindications see bronchopneumonia, page 350. In cases of very high temperature and restlessness the complete pack (10–15 minutes) is often of benefit, as is also the ice-cap to the head. This is also to be employed when cerebral symptoms are present.

In typhoid conditions, cold irrigations during the warm bath followed by rubbing are advisable. According to the advice of Jürgensen, cold and rapid spongings are of advantage whenever the arteries are flabby and not well distended. He also recommends them in attacks of weakness occurring about the time of the crisis. If the cold compresses cause prolonged coldness of the skin and cyanosis, they had better be discontinued. In these instances, a bath of short duration at about 30–32° R. (100–104° F.) is indicated.

With the onset of the crisis, all compresses and baths are to be discontinued. If after the fall of the temperature, resolution is delayed, an attempt may be made to hasten it by means of warm chest compresses.

Antipyretics may usually be dispensed with, as they often act injuriously on the heart. They are to be especially avoided immediately before the crisis. In cases of excessively high temperature, over 41° C. (106° F.), great restlessness, and cerebral symptoms, they are occasionally of transitory value. (Phenacetin 0.1–0.3 Gm. (1½–5 gr.) in the evening, or quinine by enema). Even under other conditions, internal medication may often be dispensed with. Whoever cannot desist therefrom may give an acid mixture, dilute hydrochloric acid 1–2:100 (℥ 15–30 to 3⅓ oz.). The irritating cough and the pain in the side often require the administration of codeine or morphine (added to the acid mixture), in older children also given hypodermatically in cases of severe initial pains (3 milligrams at 5 years, 5 milligrams at 10 years of age). During the febrile stage the indication for the administration of expectorants is usually absent. After the crisis they may have some claim for consideration (liq. ammon. anisat. (P. G.) whenever the secretion is abundant, ipecac in a dry catarrhal condition). Evacuation of the bowels is hastened by sugar water, fruit juices, apple sauce, and if necessary by daily enemata. In cases of obstinate constipation robust patients are to be given a laxative. Whenever cerebral symptoms are present, large doses of calomel are indicated. Otitis media with severe symptoms may require a paracentesis of the tympanic membrane.

From the beginning, the cardiac strength is to be preserved by avoidance of all unnecessary exertion on sitting up, going to stool, and in the bath; and a reserve fund should be established by a good nutritious diet.

In weak subjects it is advisable to administer mild stimulants such as bouillon, beef-tea, tea, and coffee, from the beginning, at least as soon as cardiac and systemic weakness becomes manifest; and also after each bath. These stimulants are sufficient in the majority of cases. However, as soon as the pulse becomes very frequent, small, and the artery poorly distended, together with cyanosis and dyspnœa, the administration of strong stimulants should not be delayed. Alcohol had better be avoided except in cases of unexpected collapse. In cases of weakness of the heart's action some preparation of digitalis is of service, *i.e.*, infusion of digitalis 0.15–0.8 Gm. (1½–12 gr.) according to the age, to be given in the course of two days; or, caffein. sod. benzoic in aqueous solution 0.05–0.3 Gm. (1–4½ gr.) *pro die*. It is often advisable to give digitalis first, and afterwards if it is not sufficient, caffeine. In severe cases, caffeine is also given subcutaneously with advantage; as is also camphor (spirit camphorate, ¼–1 syringeful several times daily). The heart demands careful supervision shortly before and after the crisis. In cases of severe cyanosis and dyspnœa with threatening pulmonary œdema, venesection acts apparently beneficially, provided the heart's action is still good (Baginsky, Gregor).

During the first few days after the crisis rest in bed is imperative, and should be maintained until all symptoms of consolidation have disappeared.

During the period of convalescence, the anæmia and weakness make the administration of quinine, iron, and malt preparations desirable. Later, for complete recovery and strengthening of the system, a prolonged stay in the country is advisable.

CHRONIC AND INTERSTITIAL PNEUMONIA

Etiology and Pathology.—Not infrequently, the symptoms of consolidation in bronchopneumonia last for 3–6 months or even longer. This chronic pneumonia is seen particularly after measles, influenza, and whooping-cough; less frequently after croupous pneumonia (Henoch). In the latter event, atypical forms are probably mostly responsible, and not a pneumococcus pneumonia. It consists of coagulation of the exudate in the alveoli, which are filled with cellular material that has undergone fatty degeneration, a thickening of the walls of the alveoli, and a cellular infiltration of the interstitial connective tissue. If absorption does not take place, the small celled infiltration becomes organized, and spreads, resulting in an *interstitial pneumonia*.

Otherwise, interstitial pneumonia, except a dragging pneumonia, generally develops secondarily, especially in pleurisy with thickening, and

also follows bronchitis. The same is true in connection with all kinds of affections of the lungs (tuberculosis, etc.), and in stasis of the pulmonary circulation. Jacobi believes that acute and chronic interstitial pneumonia often occur primarily, with relative frequency, and that it is not rare especially in children. The alveoli are filled with newly formed connective tissue, which later also extends into the inter-alveolar spaces and extensively infiltrates the respective portions of the lung, which subsequently become tough, grit on section, and present a bluish white appearance—induration. Later, it frequently leads to considerable shrinkage of the affected part, to formation of bronchiectasis, and to the isolation of bronchi; also to pleuritic thickening. Poor circumstances and unsanitary dwellings are favorable to the production of chronic and interstitial pneumonia. In croupous pneumonia free blood-letting (Lännec) and rapid reduction of temperature (Marchand) are held responsible.

Symptoms.—Following a pneumonia or some other of the above-mentioned diseases there are signs of consolidation in a portion of a lung (dulness, diminished breathing, bronchial breathing, fine râles, etc.). The seat is usually in one of the upper lobes, more frequently on the right side (Jacobi). There is usually fever in the beginning, often remittent, less frequently intermittent in character; later, the temperature is normal in favorable cases. Older children sometimes expectorate sputum streaked with blood. Dyspnœa, emaciation, tendency to perspiration, and sometimes diarrhœa, are present.

With the occurrence of contraction of the lung supra- and infraclavicular depressions, abnormally loud cardial pulsations, accentuation of pulmonic second sound, bronchiectases, adhesions, etc., are found. Jacobi mentions the absence of cough as being characteristic.

Resolution and *recovery* are still possible even after a year's duration, as long as contraction has not taken place, and tuberculosis is not present. Even the frequent termination in pulmonary contraction may be accompanied by a fair state of health, provided a small portion of the lung only is involved. Frequently, the subsequent phthisis, cachexia, or the results of the bronchiectasis, cause death.

The **diagnosis** often presents great difficulties, especially from pulmonary tuberculosis (examination of the sputum); also from pleuritic thickening and empyema (compare with diagnosis of these diseases).

The **treatment** should be directed towards strengthening the constitution by a nutritious diet and tonics (quinine). Woolen underwear and protection against colds are important. As remedies, the syrup of the iodide of iron and codliver oil are to be considered. Jacobi recommends arsenic and phosphorus. Later, a stay in the country, or at the seashore, and in the South during the winter, is to be urged. Pulmonary gymnastics are often useful after the fever has subsided.

EMPHYSEMA OF THE LUNGS (CHRONIC, ACUTE, AND INTERSTITIAL)

Form, Etiology and Pathogenesis.—We must distinguish three kinds of emphysema; genuine chronic, acute (pulmonary inflation), and interstitial emphysema.

1. The *chronic, alveolar, generalized emphysema* (rarefying emphysema) which runs its course with permanent enlargement of the alveoli, obliteration of alveolar septa and capillaries, loss of elasticity, which is the form so frequently seen in adults, is rarely and only exceptionally met with in older children, where it presents the same symptoms as in adults. We shall, therefore, not consider it more fully, except to mention the fact that many adults date back the beginning of their emphysema to the later period of childhood. In certain cases, a gradual development of the disease is seen in long continued bronchial asthma, adenoid vegetations, chronic, relapsing bronchitis, and in enlargement of the bronchial glands.

More frequently, a partial chronic emphysema develops in the neighborhood of portions of the lungs which have been deprived of air for some time, as in tuberculosis, and in rachitis producing deformity of the thorax, in which instance the habitual dyspnœa also assists. In these cases, *vicarious emphysema* is also indicated, which most particularly affects the sound lung in cases of contraction of an entire lung. In these cases, recovery is still possible after years, provided the original trouble disappears, since a long period of time is necessary to produce a loss of the elastic element.

2. In children, *acute alveolar emphysema* (inflammation of the lungs) is found much more frequently than genuine, chronic emphysema. The same may occur secondarily in all diseases which are accompanied by forced or labored respiration, in which instances the increased expiratory pressure is often responsible (whooping-cough, severe cough); oftentimes also the forced inspiratory breathing and the diminution of the respiratory surfaces (croup, and tracheal-bronchial stenoses, bronchiolitis, bronchopneumonia, etc.). Besides atelectases and consolidation of the lungs it also often appears "vicariously." Under these circumstances there is simply a distention of the pulmonary alveoli with the respective portion of lung. On section, these areas are pale, prominent, and do not collapse. Beyond the distention of the alveolus no histological changes are found. The anterior portions of the lungs and their borders are most frequently affected. Inflation of the anterior portions of the lungs is especially frequent in very young infants, and is almost invariably the rule in bronchiolitis and bronchopneumonia. Forced pulmonary inflation in asphyxiated newborn infants may cause a general pulmonary distention, and in rare cases may also occur in bronchial croup (Variot).

3. *Interstitial emphysema* is seen more frequently in children than in adults. It may develop as a subpleural or interlobular, but seldom as a peribronchial affection, and may extend through the mediastinum to underneath the external skin. On post-mortem examination after all kinds of diseases accompanied by severe coughing and dyspnœa, air vesicles in various stages of distention, movable and often arranged in chain-like rows, are found beneath the pleura. They are the result of rupture of the alveoli. A similar direct interlobular escape of air from small bronchi into the peribronchitic tissue is most likely to occur in localized tuberculosis. But only interstitial emphysema produces clinical symptoms after it has increased in extent and penetrated the mediastinum, usually only when it has travelled along the trachea or œsophagus and appears as a subcutaneous emphysema. Subcutaneous emphysema (originating from the lungs) is in general observed only in young infants after severe dyspnœa, and violent coughing, particularly in pertussis, as well as in diphtheria, tuberculosis, and bronchopneumonia.

The **symptoms** of chronic emphysema are sufficiently well known as they occur in the adult. It is to be noted that the barrel-shaped thorax is frequently absent in children. The acute pulmonary distention is often overshadowed by the underlying disease, and remains unnoticed. The attention is most apt to be directed to this condition when the course of the disease is prolonged, and in cases of bronchiolitis. In extensive emphysema there are found a diminution of the respiratory murmur anteriorly, prominence of the anterior portion of the thorax, together with symptoms of descent of the pulmonary margin in the right anterior mamillary line, besides a diminution of the cardiac dulness, which are unmistakable signs. After the termination of the primary condition, the pulmonary inflation recedes, and usually disappears completely even after the most obstinate attack of whooping-cough. Interstitial emphysema progresses without any symptoms. Even in cases of extensive accumulation in the mediastinum, it is only suspected when there is a rapid increase of dyspnœa and cyanosis (compression of the large vessels); but on the other hand it is easily recognized where it appears subcutaneously. There usually appears first on the neck at the base of the sternum a soft, crackling swelling, more rarely first on the cheek. Within 12–30 hours the emphysema may spread rapidly to the neck, head, lateral portions of the thorax, and the rest of the upper portion of the body. It proves rapidly fatal in most cases. When the underlying conditions are favorable, the extravasated air is absorbed within 1–2 weeks.

From what has been said the **diagnosis** is obvious. In cases of unusual size of the lungs and the corresponding depth of the pulmonary border, the readily movable margins of the lungs protect against the

erroneous assumption of a chronic emphysema. An acute inflation is usually found only on careful percussion of the pulmonary margins.

The **prognosis** is dependent upon the primary condition. The treatment of chronic emphysema is the same as in the adult, though pneumatic therapy is less frequently indicated, and respiratory gymnastics, eventually with manual aid in expiration, is sufficient. In the first place, we should seek to cure the primary disease (bronchitis, asthma). Sojourn in the country, forest air, and mountains, prove useful in these cases. Also in addition a course of treatment with arsenic and iodides. After the termination of the primary disease the acute inflammation of the lungs subsides of its own accord. In subcutaneous emphysema, capillary puncture and pressing out of the air are to be tried, in addition to relief of the dyspnœa.

INFARCTION AND ABSCESSES OF THE LUNG

The hæmorrhagic pulmonary infarction presents nothing special in the child in contrast to that of the adult.

Only the occurrence of an umbilical phlebitis from a thrombosis of the ductus Botalli, marantic thrombosis as the result of cholera infantum, may be advanced as causes. The diagnosis is rendered very difficult by the absence of sputum.

Abscess of the lung is a rare condition. It may develop in connection with protracted cases of pneumonia (comparatively frequent after influenza), foreign bodies in the lung, infectious emboli, caseous bronchial glands, or may originate from the pleura. Staphylo- or pneumococci are mostly the cause. The symptoms are usually not distinctive, and the characteristic sputum (purulent, insipid odor, elastic fibres) is often absent even after rupture through a bronchus, so that the diagnosis is rarely made during life. The abscess, which frequently causes death, is attended by symptoms of increasing cachexia, with irregular remittent fever, the cause of which is only discovered on post-mortem examination. In the case of an infant observed by me it caused an obstinate enteritis. Whenever the abscess is extensive and the dulness circumscribed, the following conditions must be differentiated: tuberculosis, bronchiectatic cavities, gangrene of the lungs, bronchopneumonia, and particularly also encapsulated, interlobular empyema, from which it is scarcely to be distinguished. Recovery by rupture through a bronchus is not rare. Exploratory puncture is useful for the establishment of a certain diagnosis.

Recently, abscess of the lungs has been successfully treated by operative measures (resection of the ribs). In most cases the pleura was found adherent. Abscesses produced by foreign bodies are mostly putrid, cause purulent pleurisy, and are not suitable for operation (Sonnenburg).

GANGRENE OF THE LUNGS

Gangrene of the lungs is comparatively less frequently observed in the infant than in the adult. It occurs, however, at all periods of life. Occurring exceedingly seldom as a primary condition, it constitutes a secondary manifestation of various diseases in weak and delicate individuals, under which circumstances it develops mostly as a result of *bronchopneumonia*. It is most frequently brought about by tuberculosis of the lungs and of the bronchial glands, measles, typhoid fever, bronchopneumonia, gastro-enteritis; next by aspiration of foreign bodies, noxious material in noma, diphtheria, ulcerative stomatitis; and again, as the result of embolism in gangrene of the skin, and of the vulva, and comparatively often in caries of the petrous portion of the temporal bone and fetid otorrhœa (Guillemot). As a sequela of croupous pneumonia it is exceedingly rare (Henoch, d' Espine). Most likely, specific, anaërobic, and septic bacteria, are to be regarded as the cause of the gangrene (Veillon and Zuber). The disease usually attacks infants in a circumscribed form; but sometimes multiple areas are found. The right lung is more frequently involved than the left.

The **symptoms** are often less marked than in adults, and the fact that gangrene is present is frequently only established on post-mortem examination. The offensive breath is often absent as is expectoration on account of the age of the child. On the other hand, children suffering from pulmonary gangrene expectorate decidedly more than under other circumstances. The expectoration presents the well-known characteristics (offensive odor, dirty grayish green, separating into three layers, and containing fibre-like shreds of lung tissue). It is frequently bloody. During the subsequent course of the disease severe hæmoptysis may take place, a condition otherwise seldom observed in children even in pulmonary tuberculosis. If the area extends even as far as the pleura, a purulent pleurisy rapidly develops, or a pyopneumothorax, the occurrence of which should always arouse a suspicion of pulmonary gangrene. Sometimes all characteristic symptoms are absent, and only the rapid prostration, the deep pallor, the weak pulse, the high remittent temperature in the course of a bronchopneumonia, lead one to suspect the nature of the disease, which almost always terminates fatally within one to three weeks. Although recovery is rare, its occurrence has been definitely established.

A definite **diagnosis** can be made only when the characteristic expectoration containing shreds of pulmonary parenchyma is present. In fetid bronchitis or in bronchiectasis the offensive odor of the expectoration is not so great, and the general condition is under all circumstances not so bad; however, pulmonary gangrene of the lungs may occur in the course of these diseases. Gangrene of lung may be readily overlooked, in cases where the existing mouth or pharyngeal affection is held

to be sufficiently responsible for the offensive odor of the breath. The local symptoms in the lungs do not as a rule furnish us with a guide towards differentiation from simple bronchopneumonia. Only very seldom do rapidly appearing cavernous signs permit us to diagnosticate the presence of a gangrenous cavity.

The **treatment** is directed towards maintaining the strength of the patient, and toward arresting the progress of the gangrene by inhalations of carbolic acid or turpentine, and by the internal administration of preparations of creosote.

ACQUIRED ATELECTASIS

Etiology.—Aside from the congenital form, atelectasis develops with relative frequency in young children, since the underlying causes are frequently found grouped together, *viz.*, general debility, occlusion of the bronchi, and insufficiency of the respiratory muscular apparatus.

In every case of bronchiolitis and bronchopneumonia, atelectatic spots of varying extent are found beside the inflamed portions. Infants suffering from active rachitis of the thorax are particularly predisposed to atelectasis, because the primary weakness of the muscular apparatus combined with the flexibility of the ribs favors respiratory insufficiency to a great extent (Hagenbach). In addition, all debilitating diseases such as typhoid fever, chronic gastro-enteritis, atrophy, etc., bring about a predisposition to this condition.

Pleuritic and pericarditic effusions, ascites, etc., lead to atelectasis by compression. The atelectasis is usually produced in occlusion of the bronchi and insufficient expectoration, by the fact that the air becomes absorbed from the isolated alveoli, causing them to collapse.

The atelectatic portions are naturally found in those situations most frequently predilected by bronchopneumonia; ascending upwards from the lower posterior portions of the lungs, and also in the anterior borders of the lungs. The individual patches vary from the size of a pea to that of a walnut, and by coalescence may even involve an entire lobe. The affected lung tissue is vascular, bluish red (like raw meat), non-crepitant, sharply defined, and depressed below the level of the adjacent portions of the lung.

The **symptoms** are rarely well marked, and are usually masked by the primary affections, especially bronchiolitis and bronchopneumonia. A clear picture of atelectasis is more readily to be found in severe rachitis and marasmus. The respiration gradually increases in frequency, and becomes superficial. Inspiratory dyspnœa and inspiratory recessions appear. The percussion note is tympanitic, and slightly diminished (usually bilateral, behind and below) over the affected portions. The respiratory murmur is diminished, more rarely bronchial; fine or crepitant râles are frequently heard in deep respiration. The fremitus may

be distinct or diminished (from occlusion of the larger bronchi). In pure atelectasis there is an absence of fever; and the temperature may even be subnormal as a result of insufficient oxidation. Whenever the atelectasis is very extensive, stasis in the pulmonary circulation results, with cyanosis and œdema of the skin. Frequently, however, fever is also present, caused by the existing primary disease. The atelectasis may gradually disappear; or by increase of the respiratory insufficiency and spreading of the bronchitis or bronchopneumonia may lead to a fatal termination.

The **diagnosis** is usually difficult, and the etiology must always be considered. A criterion is the gradual development without fever in diseases having a tendency to this condition, in addition to the above-mentioned symptoms. A differential diagnosis must be made from croupous and bronchopneumonia, pleurisy, pulmonary tuberculosis, and hypostatic congestion. Pulmonary hypostasis develops under similar circumstances and with similar symptoms in cases of greatly diminished heart action, which leads to a passive congestion of the dependent portions of the lungs. It is often difficult to distinguish it from bronchopneumonia, especially the afebrile, cachectic form of this disease.

The **prognosis** depends upon the underlying cause.

The **prophylaxis** must be directed especially to rachitis. Young infants, particularly those suffering from bronchitis, must be carefully carried about.

The **treatment** is principally directed to the underlying cause. Stagnant expectoration must be actively stimulated, if necessary, by warm baths and cold douches. The further spread of the atelectasis is to be counteracted by frequent changes of position, and by carrying the patient about.

PLEURISY

Occurrence and Etiology.—Pleurisy is less frequently met with during childhood than pneumonia; but at the same time it is a disease seen at all periods of life. It occurs in an *acute, subacute,* and *chronic,* and in a *fibrinous, serofibrinous,* and *fibrinopurulent* or *purulent,* form (empyema). The serofibrinous form is relatively rarer than in the adult. The purulent pleurisy, on the other hand, is much more frequent, and demands our principal interest since on its timely diagnosis and treatment the life of the patient often depends. From one-third to one-half of the pleuritic effusions in children are purulent; in adults only one-fifteenth to one-sixteenth (Netter). The reasons therefore we will ascertain directly.

The disease may occur at any age. Even in the newborn it is met with as a symptom of sepsis. It is not infrequent from the third to the sixth month, and from that time on it is quite extensive. The younger the child the more apt is pleurisy to appear in the purulent

form. Two-thirds of all cases of empyema (out of a total of 642 cases) were observed during the first 5 years of life, one-fourth from the sixth to the tenth year, and one-tenth from eleventh to fifteenth year. Of 145 cases of empyema 46 occurred during the first year, 30 during the second, 22 during the third, 16 during the fourth year of life (Netter). From the sixth to seventh year and upwards the serofibrinous effusions predominate.*

Boys are more often affected than girls. This depends on the fact that croupous pneumonia, the most important cause of pleurisy, is more frequent in boys. The frequent connection with croupous pneumonia may be largely influenced by the prevailing occurrence of pleurisy between the months of January and May. As accessory factors colds are sometimes mentioned, less frequently trauma.

Pleurisy often occurs as a primary disease. The majority of these cases depend upon pneumococci, in which no doubt the primary pneumonic focus may occasionally have been overlooked. Nevertheless, primary pneumococcic pleurisies undoubtedly do occur, just as these bacteria may cause primary arthritis or peritonitis. According to the experiments of Arnold and Grawitz with inhalations of dust one can readily understand how the pneumococci may migrate through the lungs into the pleural cavity, at the same time allowing the lungs to remain intact.

In the majority of cases, pleurisy appears as a secondary affection. Most frequently in affections of the lungs more than elsewhere, in croupous pneumonia (metapneumonia), then in bronchopneumonia, bronchitis, gangrene of the lungs, etc. Pleurisy is met with as a result of pulmonary tuberculosis less frequently than in adults, and is mostly fibrinous or serofibrinous in character, and often simulating the primary form. Then again the cause may depend upon ulcerations and inflammations of neighboring organs (bronchial glands, pericarditis, caries of the ribs and vertebræ, peritonitis, and especially perityphlitis (Wolbrecht), or upon acute infectious diseases, especially in scarlet fever, then in angina (Grober), diphtheria, grippe, measles, whooping-cough, typhoid fever, erysipelas, variola, acute rheumatism, intestinal or general sepsis, acute osteomyelitis, etc., as well as in nephritis and syphilis.

Bacteriology.—During recent years the bacteriology of pleurisy has been much advanced. In the fibrinous and serous forms, bacteria are more frequently found in the culture test than was formerly supposed, though often in such small numbers that they may be regarded as being partly accidental and not as causative factors. Generally, pneumococci are present: less frequently, staphylo- and streptococci, or tubercle bacilli. Schkarin also constantly found micro-organisms in the serous

* These figures are only of relative value since they refer to hospital cases only. Mild cases are more apt to be treated at home than severe cases, which are generally cases of empyema.

or serofibrinous pleurisies of nurslings, pneumococcus in pure culture in two-thirds of the cases. Effusions which are apparently free from bacteria are often tuberculous in character, and by inoculation of animals tubercle bacilli are usually demonstrable.

On the other hand, numerous pathogenic germs are found to be the cause of empyema, the pneumococcus being most predominant. Netter found in one-half to one-fourth of the cases pneumococci in pure culture, besides pneumococci associated with strepto- and staphylococci, tubercle bacilli, then streptococci in pure culture in 13–16 per cent. of cases, less frequently with staphylococci and tubercle bacilli. Tubercle bacilli were found in 5–7 per cent. of the cases, mostly as a mixed infection; furthermore, staphylococci and coli bacteria, but only in isolated numbers in pure culture; and lastly septic bacteria in putrid empyema.

In contrast to the adult, Netter found a remarkable difference in the participation of the various micro-organisms, viz:

	Pneumococci	Streptococci	Tubercle bacilli
Adults (154 cases).....	24.9%	41.2%	17.6%
Children (90 & 81 cases)..	80.7–65.4%*	13.3–19.7%†	5.5–7.4%

Accordingly, in infantile empyema the pneumococcus is most predominant; whereas on the other hand the streptococcus is the most frequent cause of empyema in the adult. The investigations of other observers verify the predominant position of the pneumococci in empyema of children. Koplik (72 cases) found the pneumococcus in pure culture in 60 per cent., streptococcus in 15 per cent., staphylococcus in 9 per cent. of cases. At the highest 7 per cent. were tuberculous. Beck found pneumococci in 75 per cent.; Blaker in 94 per cent. of cases of infantile empyema. Whenever the pus of an empyema appears bacteriologically sterile, it indicates a tuberculous nature. From what has been stated the preponderance of purulent pleurisy in the infant in contrast to the adult is clearly established. It depends partly on the fact of the great tendency to suppuration of the pneumococcus pleurisy of infancy. On the other hand, tuberculous pleurisy in any form is not frequent in children, whereas in adults it embraces one-fifth of the cases of empyema and fully one-half of serous pleurisy.

The character of the empyema may be assumed with considerable certainty from the very beginning. In primary cases and following croupous pneumonia it is commonly a pneumococcus empyema, as also often in bronchopneumonia and grippe. In the acute infectious diseases streptococci preponderate, more rarely staphylococci, especially in scarlet fever, diphtheria, measles; and in addition the bacteria of the underlying disease (typhoid, tubercle bacilli, coli bacteria) may be found. Most commonly the micro-organisms penetrate the pleural cavity di-

* In pure culture 73.8 per cent. and 56.8 per cent. respectively.
† In pure culture 10 per cent. and 16 per cent. respectively.

rectly from the lungs, or from some other disease focus in the vicinity; and frequently from the peritoneum or tonsils through the blood and lymph channels.

Pathological Anatomy.—A loss of lustre and adhesions of the pleural reflections are found in many cases on post-mortem examination, in which no definite symptoms were ever present during life. In the mildest form, the dry or fibrinous pleurisy is a frequent accompaniment of various pulmonary diseases, often without any clinical manifestations. There is a lack of lustre and injection of pleural surface to a varying extent. The surface is rough and covered by a felt-like layer of fibrin. In pleurisy with effusion the pleural cavity is filled with a serous fluid, mostly of a yellowish color, seldom bloody, at times clear, sometimes cloudy, containing shreds of fibrin which are also found on the pleural surface as a felt-like covering. If the lymph-cells found in addition to the endothelial cells in the effusion are very numerous, a seropurulent or a complete purulent exudate will be formed, which occasionally though rarely is putrid in character. In slight effusions the exudate becomes fibrinopurulent in character from deposits of thick villous fibrin (see Plate 48.) The pleural layers are often very much thickened.

Complete restitution ad integrum may take place from absorption of the inflammatory exudate. After the termination of a pleurisy, however, either card-like or extensive adhesions of the surfaces of the costal and pulmonary pleura often remain; and often extensive thickening of the connective tissue (pleuritic thickening) which may be from 1–2 cm. in thickness, going hand in hand with retraction of the affected half of the chest, shrinkage of the lung, and bronchiectasis. Large, purulent effusions do not become absorbed, but when untreated often lead to a fatal termination from cachexia or pyæmia, or they may rupture through the bronchi or chest wall (empyema necessitatis). Regarding the bacterial content see above.

General Course.—Except the physical finding, the symptoms of pleurisy are often varied and changeable according to the form of the disease met with, whether acute or chronic, serofibrinous or purulent, primary or secondary, so that it is scarcely possible to give an adequate clinical picture. We shall therefore be content to point out several of the most salient features.

In acute cases, the beginning of the disease often manifests itself by symptoms appearing in rapid succession. The patient is attacked in the best of health by malaise, chilliness, fever, and headache. Vomiting frequently occurs, though not as often as in croupous pneumonia. In some cases during the first years of life convulsions and somnolence occur, whenever the disease is ushered in with a high temperature. From the beginning, respiration is painful, increased, suppressed, and superficial. Older children complain of a very painful spot in the side;

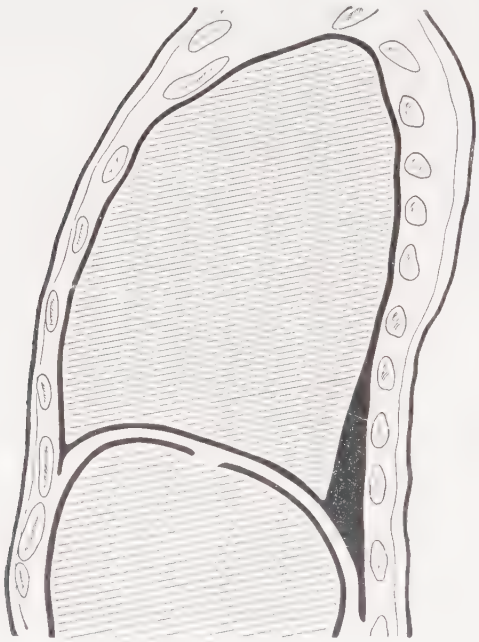
younger children refer the pain to the epigastric region. The pain may be much more severe than in pneumonia. A painful, dry, short, and very harassing cough often sets in, but may also be absent. Examination often reveals on the first or second day the presence of a fibrinous pleurisy by friction sounds, diminished breathing, and local pain on pressure over some point of the pulmonary surface. Cases of acute fibrinous pleurisy of this kind often recover in a short time, with a rapid abatement of the fever and other symptoms. They do not present anything special, and will therefore not be considered any further.

Whenever a pleuritic effusion has taken place the clinical picture is different. This condition is often demonstrable as early as the second or third day, usually, however, only later. The following symptoms then develop according to the size of the effusion and the rapidity with which it accumulates. The child avoids unnecessary motion and prefers to be on the affected side in order to use the sound lung for breathing. For this reason, nurslings often will only drink from one breast, for instance, in right-sided effusions from the left breast. The *expression of the face* is anxious, and painfully distorted in coughing or crying. The lips and cheeks are pale. The breathing is still rapid and superficial, but expiration is often not as suppressed and grunting because the pain usually subsides with the appearance of the effusion. The sound side breathes more deeply than the affected one. Dyspnœa makes itself manifest while speaking or making any movement, and increases with the amount of the effusion, and may be accompanied with cyanosis in case of considerable displacement of the heart. Inspiratory recessions of the lower aperture of the thorax are less common than in pneumonia. In large effusions they are more apt to affect the sound side. The cough frequently disappears while the effusion is taking place, but may also continue and be spasmodic in character. It is frequently followed by suppressed crying or whining. The patients avoid loud crying. Many times they do not seem to suffer any pain, so that in the absence of cough there is nothing to direct the attention to the chest, leaving the general indisposition to control the clinical picture—fever to a more or less degree, malaise, coated tongue, poor appetite, disturbed sleep, restlessness, increased pallor. The amount of urine is diminished, especially in rapidly increasing effusions, albuminuria sometimes occurring if the temperature is high. Often there is constipation. Under these circumstances, only a careful and systematic examination will guard against overlooking a "latent pleurisy," or perhaps even the assumption of "dentition fever."

In effusions of an appreciable degree the physical examination reveals very important signs. *Inspection* shows an obviously diminished excursion and dragging of the affected side, besides the accelerated, superficial, and, when pain is present, jerky respiration. In very exten-

sive effusions severe dyspnœa and expiratory distention of the veins of the neck are seen. The affected side may be almost motionless. Obliteration of the intercostal spaces is rare in acute cases, and may even be absent in purulent cases, contrary to a widely accepted opinion. On the other hand, one can readily recognize with the unaided eye, distention of the affected half of the chest, in extensive effusion. This is especially plain in front below the clavicle, and when the patient is observed from behind while in a sitting posture. Often there is also elevation of the shoulder. This distention of from 2–4 cm. may be easily established with the tape measure. The frequent use of the tape measure is to be recommended, because the increase and decrease of the effusion may thus be determined. Even after the disappearance of the fever, the accelerated superficial breathing is still conspicuous. A diminution in the size of the affected side of the chest, with descent of the shoulder and standing off of the scapula, is often found after absorption of the effusion.

Fig. 77.



Beginning pleurisy with slight effusion behind and below (the effusion is colored a deep black). Slight dulness and diminished breathing. (Schematic sagittal section through the left half of the thorax).

Palpation commonly reveals sensibility to pressure, especially if it is made between the intercostal spaces even in children who otherwise do not complain. This is in favor of pleurisy in so far as there is no active rachitis of the ribs. In large effusions a displacement of the apex beat and descent of the lower hepatic border are often to be felt.

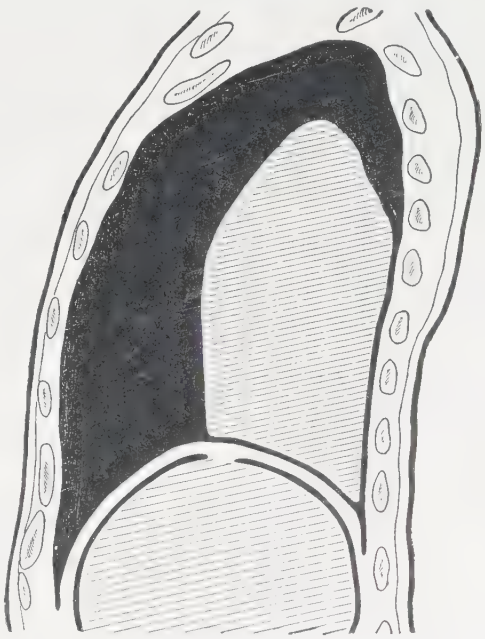
Percussion is the most important part of the examination in pleurisy with effusion. It permits of a much more certain judgment than auscultation, and is best carried out with the patient in an upright position. Percussion over the affected pleura is often painful. At first a slight dulness over one lung behind and low down almost always shows itself. One must percuss lightly, otherwise the percussion note elicited by a thin layer of fluid will be drowned by the co-vibrations of the lungs (see Fig. 77).

With the increase of the effusion, the dulness gradually extends further upwards and spreads out toward the side and to the front, taking in Traube's space on the left and generally diminishing behind above

to below in front. Should an effusion develop where adhesions of the pleural folds already exist, the lung may be held fast behind and below, and the effusion will then accumulate more above, laterally and in front, producing a corresponding dulness (see Fig. 78).

The feel of great resistance encountered by the percussing finger is very characteristic. On account of the thin elastic chest wall, it is much more marked in the child than in the adult. It alone often permits a diagnosis of pleurisy. The dulness diminishes near the upper border of the effusion, where it is tympanitic. In younger children with large effusions the note is never as flat as in adults, because the underlying lung still makes itself manifest as long as it is not completely compressed. In a very large effusion, which reaches the third rib anteriorly, the note is very tympanitic and often hyper-resonant beneath the clavicle. Among others, Rauchfuss has called attention to an important sign of dulness. In a pleuritic effusion on the one side, which as a rule extends as far as the vertebral column behind, there is also often found on the sound side adjacent to the vertebral column a striplike area of dulness in the form of a small triangle. The apex of this triangle reaches almost as high as the dulness on the affected side, and its base, 2–5 cm. in breadth, passes over into the liver dulness below.

Fig. 78.



Very large effusion (colored black) filling the pleural cavity in front (also Traube's space) and the upper portion posteriorly. The unusual absence of effusion in the posterior lower portion is explained by old pleuritic adhesions which bind down the lung in this situation. Schematic sagittal section through the left half of the thorax.

The *displacement of adjacent organs* by large effusions is almost pathognomonic of pleurisy. In left-sided effusions, the heart is pushed to the right very early, the apex beat being displaced to the right of the sternum, and the cardiac dulness as far as the right mammary line. In right-sided effusions the heart may move to the middle axillary line. The descent of the liver is very significant in right-sided effusions, but the depth and unequal position of the lower border of the liver in the child during health must be taken into consideration. In an effusion filling an entire side, the dulness will extend to the distant border of the sternum anteriorly,—an important sign.

In pleuritic effusions *auscultation* is of less value in diagnosis than

percussion, and may readily lead to errors, because the respiratory murmur is often almost normal even in not inconsiderable effusions. Of course in a large effusion the vesicular breathing is usually diminished or absent. On the other hand, it is quite frequently the case that distinct bronchial breathing and bronchophony are heard over an effusion in acute cases, and sound as if coming from a distance. The bronchial breathing disappears only after some time, if a large effusion leads to compression of the lung. Fremitus is, of course, always diminished or abolished over the area of an effusion; but it is often difficult to prove. Ægophony is seldom heard in younger children.*

Pleuritic friction is heard less often in the beginning of a pleuritic effusion than during absorption above the boundary of the effusion. Crepitant râles are, at times, over the upper border of the dulness during inspiration (unfolding râles). Tinkling râles are not infrequent; and in isolated cases almost amphoric breathing is heard to the left over the stomach.

If the physical examination has demonstrated the presence of a pleuritic effusion, the physician is confronted by the important question whether he is dealing with a serofibrinous or a purulent (empyema) effusion. Only rarely is differentiation possible from the local finding, much more from the origin and the whole course of the disease. One can say, therefore, with some truth that serofibrinous and purulent pleurisy are two very different diseases as to their origin, symptoms, course, prognosis, and treatment (Barthez and Sanné). And certainly it is not proper to regard an empyema as a mere reinforcement and an advanced stage of a serofibrinous pleurisy, but one must agree to a great extent with those (Dieulafoy, Netter, and others) who say that an acute pleuritis is destined from the beginning to be or not to be purulent. A short and separate consideration of the two forms is therefore apropos here. If we have not yet made this distinction clear, it was for the purpose of avoiding repetition, and because the physician learns to distinguish the two forms only during the course of the disease.

1. As mentioned under etiology, *serofibrinous pleurisy* rather infrequently attacks children under five years of age, but is also seen during the nursing period. Occurring primarily, it is often ushered in as rapidly and violently as has been described. However, it often also appears secondarily, most frequently after croupous and bronchopneumonia, or acute rheumatism, and then after acute infectious diseases (measles, scarlet fever). It frequently begins inconspicuously and stealthily without any material local symptoms. The physician is consulted because the child is becoming pale, thin, tired, and is without appetite, or because recovery does not seem to take place after an acute disease. Exami-

* Pitres has called attention to a sign which is but little known. If the outside of the affected half of the thorax is percussed with a superimposed coin on a second coin, a metallic sound (*signe du sou*) is heard on the other side in the presence of an effusion. The second pulmonic sound is often accentuated from the impaired pulmonary circulation. Otherwise the heart sounds are diminished by the effusion.

nation then usually reveals a considerable effusion. In general, the course is rapid and mostly terminates after 3–4 weeks, or at least after 2–3 months. Even in cases where the initial symptoms were severe and associated with high fever, the pain, temperature, and effusion, subside as early as 1–2 weeks, though not as rapidly as in croupous pneumonia. Acute pleuritis seems to be unusual, and sudden deaths are very rare occurrences. Quite frequently the effusion is hæmorrhagic in small children without being tuberculous, as in influenza, acute rheumatism, hæmorrhagic diathesis. The so-called *rheumatic pleurisy* is frequent in children of from 10–15 years of age. It often originates from an unknown cause, rarely after taking cold, more frequently after angina and acute articular rheumatism in which it is prone to appear bilaterally. The effusion is frequently free from bacteria.

Tuberculous pleurisy is much rarer in children than in adults. It often develops slowly and stealthily. The mononuclear lymphocytes are supposed to predominate (see tuberculosis).

During the first days, the *temperature* in serofibrinous pleurisy is usually pretty high, 39–40° C. (102–104° F.), and then during about the 2nd–3rd week, with an effusion remaining stationary, is apt to assume a remittent, descending, type, and to disappear very gradually during absorption.

The *pulse* is very much accelerated during the febrile period. With displacement and compression of the heart and large trunks of vessels, it becomes small and weak, sometimes irregular. In more extensive effusions it may also be accelerated without fever, and bounds rapidly in height especially after slight exertion or motion.

Complications which are not caused by some other underlying affection are rarely observed. Simultaneous peritonitis or pericarditis mostly depend on a common cause (tuberculosis, acute rheumatism).

Recovery in acute cases is usually complete, often, however, leaving adhesions of the pleural folds, which may produce diminished mobility of the lungs lasting for years or remaining permanent. Absorption frequently takes place rapidly within 2–3 weeks and is accompanied by increased diuresis, though it often lasts very much longer. If the effusion becomes very large and is not removed in time, it may cause death from mechanical pressure on the heart and large vessels amid cyanosis and pulmonary œdema, or by cardiac thrombosis and embolism of a pulmonary artery. If the effusion is absorbed slowly, a thickening of the connective tissue of the pleura (pleuritic thickening) often remains for many years, and this usually continues to produce dulness and diminished breathing below posteriorly. In prolonged and chronic cases, especially where a large effusion has been permitted to undergo absorption for a long time without the aid of medical skill, the lung which is bound down for months by the thickened and shrinking pleura never

becomes fully expanded. A diminution of the respective half of the chest results (rétrécissement), with a narrowing of the intercostal spaces, scoliosis, and approximation of the scapula to the vertebral column. At the same time the heart and the liver are drawn towards the contracting pleural cavity. The sound lung presents vicarious emphysema. Even after many years' duration, retrogression of the rétrécissement, pleuritic thickening, and expansion of the lung, are still possible in children under favorable conditions. Otherwise, a chronic interstitial pneumonia with bronchiectasis frequently develops.

2. *Purulent pleurisy* (empyema) plays a much more important rôle in practice among children than serous pleurisy. The two forms are scarcely to be distinguished by physical examination, since the œdema of the thoracic wall which is often mentioned as point of differentiation is also usually absent in empyema. On the other hand, the origin and course of the disease frequently places us in a position to recognize with great certainty the purulent nature of the pleurisy. Very often the effusion is purulent from the beginning, as in pyæmia, gangrene of the lungs, scarlet fever, and croupous pneumonia occurring during the nursling age. The transition of a serous effusion to an empyema may take place within a few days, and the large bacterial and lymphocyte content in a recent effusion (for instance, during a croupous pneumonia) will often permit us to foresee this transition. A purulent pleurisy usually presents more violent symptoms than the serous variety. The effusion increases rapidly and may soon involve one side entirely. The discomforts are greater; palpation and percussion are often more painful. The general condition is much more disturbed. Within a short time, there is a loss of strength and loss of appetite, pallor and anæmia and emaciation. Sweats appear. Bilateral empyema is not altogether rare. Absorption of the effusion may fail to take place even within 4–6 weeks. In long continuance, enlargement of the liver and spleen sometimes occurs.

The temperature is higher on the average than in the serous type, and may attain 40.5–41.5° C. (105–107° F.) in the beginning. It may be drawn out for weeks, may vary in height, and may often be intermittent. Very frequently, especially in the later course of the disease, fever may be entirely absent. In striking contrast to the afebrile condition is the greatly accelerated and small pulse, which is markedly increased on sitting up. Without the life-saving treatment, the disease often leads to chronic invalidism and death, amyloid degeneration, purulent metastases, rétrécissement (contractures), or to rupture of the pus externally or through the bronchi. Heubner describes a multiple, purulent inflammation of the serous membranes.

Chronic empyema is frequently unrecognized, and may for instance be mistaken for caseous pneumonia. It often differs from this, however,

by the displacement of the neighboring organs and eventually by rétrécissement and diminished fremitus. The great importance of empyema during childhood makes desirable a short review of the most important forms which have been studied, especially by Netter.

(*a*) As stated under etiology, the *pneumococcus empyema* is by far the most frequent form. It may occur primarily or secondarily, most frequently as a sequela of croupous pneumonia after many days or perhaps even weeks (metapneumonia empyema).

Should the temperature in a croupous pneumonia abate gradually in order to soon rise again, should a high temperature set in again a few days after a typical crisis, or should the decline of temperature remain absent for ten or more days, the possibility of the presence of an empyema must be considered, particularly if the patient is less than four to five years of age. With comparative frequency empyema begins before the crisis (parapneumonic empyema).

With the advent of a pleurisy in an existing pneumonia, dulness is increased, fremitus is diminished, and bronchial breathing and bronchophony are usually still more increased. It is peculiar that at certain times empyema complicates pneumonia only very rarely, at other times again more frequently. Netter found the fever of a pneumococcic empyema more frequently continuous than intermittent. Fever, however, may be entirely absent, according to Würtz, even in half of the cases which received hospital treatment. The disease may quite frequently run a "latent" course, *i.e.*, the patient scarcely complains of pain and dyspnœa and cough are slight. Not altogether infrequently a bilateral empyema may be present. At times the effusion leaves the lower portion of the pleural cavity free, and may be situated only over an upper lobe, or anywhere in the middle of the lung, or even may be interlobular, conditions which must be carefully considered from a diagnostic point of view. The effusion is opaque from the very beginning, and very rapidly becomes purulent. It frequently contains in large masses coarse shreds of fibrin almost as thick as a finger.

The pneumococci pus often presents a characteristic appearance. It is thick, slimy, greenish, has an insipid odor, and leaves no sediment on standing. The pneumococci in the pus are arranged in long chains, and are distinctly lancet-shaped. Sometimes the effusion becomes absorbed spontaneously, if it is only small in quantity. Sometimes it ruptures through the bronchi and appears in mouthfuls in older children as a purulent sputum. In such cases there is often no pneumothorax, probably because the communicating opening is very small. An empyema necessitatis often occurs, mostly through the anterior wall, and manifests itself by œdema and fluctuating swelling in from the third to the fifth intercostal space. The purulent inflammation at times also involves the pericardium, especially in left-sided empyema or may lead

to pulmonary abscess, metastases, in the form of peritonitis, osteomyelitis, arthritis, and skin abscesses (Hagenbach-Burckhardt). Meningitis is not seldom observed, and is most apt to occur in cases of protracted course.

The **prognosis** in pneumococcic empyema is comparatively good. Most cases recover with timely operative evacuation of pus. During the first two years, the prognosis is more dubious. During early years serious complications, bronchopneumonia, purulent pericarditis, and meningitis, are more apt to occur, and probably always terminate fatally. In isolated cases, a simple puncture may suffice to bring about recovery.

(*b*) *Streptococcic empyema* is frequently found in scarlet fever, measles, angina, and erysipelas. It develops very rapidly with high fever, typhoid symptoms, and rapid prostration. Diarrhœa is frequently present. The effusion ascends to the top in the shortest time, and immediately reaccumulates after evacuation. The pus is thin and shows on standing a grayish deposit covered by a large quantity of serous fluid. Pericarditis, sometimes peritonitis, and very frequently general septicæmia, are important complications. The prognosis is bad; Netter lost seven out of nine cases operated on. As treatment he recommends thoracotomy and irrigation with boiled water.

(*c*) *Tuberculous empyema* is quite rare. It usually develops slowly and chronically without any material discomfort, and often occurs as a sequel to a serous effusion. It may lead to pyopneumothorax and to pulsating empyema in left-sided cases. The diagnosis is based on the existence of tuberculosis in one of the lungs or elsewhere. The effusion is often seropurulent, sometimes fetid from the beginning, or becomes fetid after operation. The pus may also become thick and greenish in color, from the pneumococcic content. It is often difficult to distinguish the empyema of tuberculosis patients from tuberculous empyema (compare with the chapter on tuberculosis).

(*d*) *Fetid empyema* depends upon a previous or on an existing communication of the pleural cavity with the external air which has afforded entrance to septic germs (pulmonary gangrene, etc.). The picture of pyopneumothorax is produced by the formation of gas in the pus, which aids in diagnosis in so far as a tuberculosis pneumothorax can be excluded. The evil sometimes develops after typhoid fever, measles, or chronic otitis. As treatment, thoracotomy or resection of the ribs with irrigation are to be employed.

The **diagnosis** of pleurisy often presents difficulties in the child which are absent in the adult. Dry pleurisy is easily recognized as soon as pleuritic friction sounds are to be heard; these are, however, more frequently absent than in the adult. Soft friction sounds may present similarity to crepitant râles. Crepitant râles are heard only on inspira-

tion with one breath, are uniform, and often change after coughing. The isolated friction sounds are more apt to be less uniform, and are also heard on expiration, and have a tendency to appear over a circumscribed area. Pleurisy with effusion in older children is as easily diagnosed as in adults. The latency of pleurisy is not based on the nature of the disease but on the carelessness of physicians (Henoch). The great resistance of the dulness, the diminished or abolished fremitus, the bronchial breathing heard only softly, the accelerated, superficial, and in the beginning painful breathing, the expansion, and the very much impeded movements of the affected half of the thorax, or the displacement of the neighboring organs, render the diagnosis easy.

The recognition of the disease in young children is often very difficult. Here, the existence, for instance, of a moderate dulness will often cause us for a long time to be uncertain whether a pleuritic effusion or pulmonic consolidation is present. Should the child not favor us by coughing or crying, it is often impossible in the youngest subjects to test the fremitus, the constant diminution of which constitutes one of the most important signs of an effusion. Furthermore, in children distinct bronchial breathing and bronchophony are often heard over a recent effusion even of considerable degree. Finally, beside the pleurisy, bronchitic sounds, at times even amphoric breathing, are heard with relative frequency. In these difficult cases with indistinct fremitus and bronchial breathing the peculiar resistance of the pleuritic dulness can alone apart from an exploratory puncture render the diagnosis possible at the beginning in small effusions. Otherwise, we must take into consideration the remaining symptoms against those diseases in question. In this respect croupous pneumonia, the broncho- and tuberculous pneumonia are the most important. The differential diagnosis of these has been considered in the respective chapters. Later on, the diagnosis is much facilitated by the observation of the course and the typical physical signs, and also by the manner of the increase and diminution of the dulness, and by the obliteration of the intercostal spaces, the paravertebral dulness, the deficient displacement of the pulmonary borders, and the total filling up of the complimental spaces. For diagnosis, radioscopy may also be utilized which will show the displacement of the neighboring organs particularly well.

Hydrothorax always develops in conjunction with other transudations, almost always bilaterally, and is afebrile, without pain and without cough. Its level is changed by change of position. The rare echinococcus cysts and tumors of the pleura and lungs are very difficult to diagnose.

If the diagnosis of a pleuritic effusion seems to be established, the important question to be decided is whether a serous or purulent effusion is present. In the above description of the disease those points in

favor of a purulent or non-purulent nature of the effusion are sufficiently brought out. To the great detriment of the patient empyema is often unrecognized when it runs an afebrile or subfebrile course and bronchial breathing is present over the effusion, both kind of cases occurring very frequently.

Pus is probably present whenever the patient in question is less than five years of age, if the pleurisy has joined a croupous pneumonia and the fluid rapidly fills the entire half of the chest. A serous effusion is almost always present whenever the patient is over ten years of age, or if the pleurisy has become associated with pulmonary tuberculosis or acute rheumatism. In order to determine, exploratory puncture is to be done (see below). An encapsulated empyema in an unusual situation renders recognition difficult, and may also give rise to confusion with pericarditis.

Often pleuritic thickening cannot be distinguished from a fluid exudate because the same may also cause resistant dulness and diminished vesicular breathing. Fremitus frequently reappears in pleuritic thickening. The diagnosis is generally made certain by the course and narrowing of the affected side; frequently, however, only by an exploratory puncture.

Exploratory puncture is to be undertaken where an extensive dulness does not permit the decision whether a consolidation of the lung or a serous effusion is present, and where the longer duration of this dulness might make evacuation desirable in case of an effusion. Exploratory puncture should always be done as soon as well grounded suspicion of empyema is present, since it demands the immediate removal of the pus. In practice infantile empyema frequently remains unrecognized for weeks. For this reason children's hospitals often have the experience of receiving cases only when they are in a desperate condition. By timely exploratory puncture and operation good health could again be given to many a child that perishes miserably from exhaustion, metastases, etc., the result of an unrecognized empyema. Very frequently, the exploratory puncture is the only measure to distinguish small effusions from pulmonary consolidation; the splenopneumonia of Grancher is scarcely to be recognized in any other way.

With antiseptic precautions exploratory puncture is entirely without danger. The probability of sometimes infecting a healthy pleura by puncture of a bronchiectatic or tuberculous cavity is extremely slight. The Pravatz syringe must not be employed for puncture because the pus and shreds are often so thick in metapneumonic empyema as not to enter a fine cannula, leading the physician to believe that he is not dealing with an effusion. For this reason one must use a larger and well fitting syringe, the cannula of which has a diameter of one millimetre. A spot at which dulness is the greatest and where fremitus and

increased breathing if possible is absent is chosen for puncture. The child is held firmly in the sitting position by the mother. For instance the position on the table as shown in Fig. 79 is a favorable one. With the index finger of one hand the intercostal space is carefully found in order not to encounter a rib with the needle, the skin having been disinfected. The needle is plunged in to the depth of about 1–2 cm. If on withdrawal of the piston no fluid is obtained the needle is pushed in somewhat deeper. The puncture is closed by adhesive plaster. The aspirated fluid permits the unaided eye to determine the diagnosis, not only whether a serous or purulent pleurisy is being dealt with, but often also what is the nature of the empyema (see above). The exact diagnosis

Fig. 79.

Exploratory puncture of the pleura. Child sitting on the edge of a table.

is made with cover-glass preparation respectively by culture and eventually by inoculation. The serous effusion has a specific gravity of 1.016 to 1.024; one drop of acetic acid produces opacity and a flocculent sediment, in contrast to the transudation in hydrothorax which also has a lower specific gravity. In a smaller, sacculated, or interlobular, empyema with extensive thickening of the pleura the first puncture often does not yield any pus. A second puncture is then made at another point, and is repeated if necessary after several days. It is well to point out to the relatives from the start the possibility of a negative puncture. Considerable pleuritic thickening is manifested by the greater resistance encountered in the introduction of the needle.

The **prognosis** in pleurisy is on the average much better than in adults. Serofibrinous pleurisy usually terminates in complete recovery in a comparatively short time, except when it is of tuberculous origin. If a larger exudate lasts for a long time without being removed by medical skill, lasting contracture may remain. In individual cases temperature, pulse, and dyspnœa, are criterions, and the tape measure indicates the increase and decrease of the effusion. Empyema also furnishes a better prognosis than in the adult because it mostly depends on pneumococci, and the pneumococcus pneumonia offers the best chances and almost always terminates in complete recovery, if operation is performed sufficiently early. Other empyemæ, especially those dependent upon streptococci, have a much more unfavorable prognosis. The bacteriological examination of the pus may therefore be utilized for prognostic purposes.

Prophylaxis consists in prevention, *i.e.*, the best possible treatment of the causative diseases. Furthermore, in the care and hardening of the respiratory passages and the lungs, and in the watchful care of the mouth and throat.

Treatment.—An attempt may be made to abort a very recent and isolated fibrinous pleurisy by diaphoresis. The patient is kept in bed and on a fluid diet as long as he has temperature. In more severe cases, the application of overlapping strips of adhesive plaster, producing pressure and covering the diseased spot and its vicinity, will be found useful for the purpose of fixation of the affected side and for the relief of pain. The application of iodine to the affected side is a favorite measure. It is rarely necessary to administer codeine or morphine even for a short time on account of the severe pain. (See above in the treatment of croupous pneumonia.)

Whenever a pleuritic effusion has taken place, rest in bed must be advised even in cases of small effusion and absence of fever. Sitting up and all unnecessary exertion are to be avoided as much as possible. As long as temperature is present a fever diet is indicated (compare with bronchopneumonia). It is perhaps advantageous to curtail the ingestion of water during the stage of effusion. A nutritious diet consisting of an abundance of eggs, meat, beef juice, butter, extract of malt, etc., is in place in a febrile case, and particularly in cases running a prolonged course. One seeks to stimulate the appetite and improve the nutrition of the patient.

As long as high temperature exists, cold chest compresses changed every two hours are generally useful; the patient in this case, however, should be guarded against active motion and exertion. In older children in cases of high temperature and severe pain, an ice cap may be applied for relief in the beginning; and, if necessary, cold applications to the head. When the fever subsides and in cases of prolonged course warm chest compresses are indicated to facilitate absorption. Many physi-

cians apply ointments locally, although their value has never been proved (ung. potass. iodide, iodine, or salicylic-vasogen). They had better be used only after the termination of the effusion, at which time inunctions of green soap act well.

Of the internal remedies salicylates often prove of decided value for absorption (sod. salicyl., 2.0–8.0 Gm.: 150.0 c.c. (30–120 gr.–5 oz. diluent) 5 times daily, but better 0.25–0.75 Gm. (4–12 gr.) aspirin in sugar water, 3 times daily, not only in pleurisy based on a true acute articular rheumatism, but also in forms of unknown cause. In large effusions, absorption is stimulated by means of diuretics; for instance, infus. digitalis 0.2–1.0 Gm.: 100 c.c. (3–15 gr.: 3½ oz.) if necessary with addition of 2.0–5.0 Gm. (15–75 gr.) of diuretin 10 Gm. (2½ dr.) 3 times daily. Decoct. cort. chinæ repeated several times acts as a stimulant to absorption and to the appetite. Camphor or caffeine are employed subcutaneously in weakness and collapse. Rest in bed is to be maintained until complete absorption of the effusion has taken place. In protracted cases sitting up in bed may be cautiously allowed, even when small amounts of effusion are still present; but fever must be absent.

In dragging cases with anæmia, preparations of cinchona or the syrup of the iodide of iron, malt extract with iron, or ferrous iodide, levico water and codliver oil during the winter, are advisable in addition to warm chest compresses. Remaining out of doors as much as possible in the fresh air and sunshine with little or no exercise, and brine or sea-salt baths are to be recommended. Fango packs are also to be considered, and, furthermore, in chronic cases a sojourn in the country, or in a warm climate during the winter, is to be recommended.

Recently good results are reported from injections of thiosinamin and fibrolysin in cases of resulting adhesions and pleuritic thickening (Friedländer, Mendel). According to the splendid results observed even in interstitial inflammation and cicatrization this method of treatment indeed deserves notice. General and pulmonary hygiene, also cornet playing, massage as well as gymnastics, which may be carried out in connection with salt-water baths, as for instance in Kissingen, Ems, Reichenhall, and sojourn in the mountains are the best methods of preventing contractures and imperfect expansion of the lungs after the termination of a pleurisy.

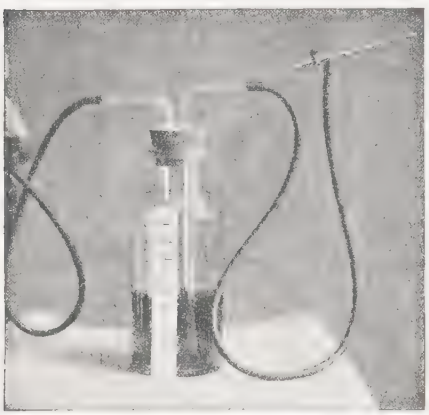
THORACENTESIS—PUNCTURE OF A SEROUS EFFUSION

The removal of the effusion in children is less frequently necessary than in adults, since absorption is more apt to occur early and spontaneously. Otherwise the indications are of the same value. In rare cases puncture becomes in the beginning of the disease a means of preventing death whenever the effusion by its rapid increase causes dyspnœa, cyanosis, and cardiac weakness; then, again, in very large effu-

sions which lead to displacement of the heart and severe dyspnœa in the average patient, when the effusion has risen to about the level of the third rib anteriorly and the middle of the scapula posteriorly. Whenever possible, however, one should wait until the fever has subsided (about the third week), since otherwise the effusion is often rapidly renewed. Finally, paracentesis is also indicated whenever a smaller effusion shows no tendency to spontaneous absorption after abatement of the temperature. No fixed rule can be laid down in these cases. If an effusion has existed for a longer period than 6–8 weeks, thickening, adhesions, and impeded expansion of the lungs usually result.

Method of Puncture.—A trocar of at least 2 mm. calibre and if possible having a lateral return flow is selected (as in Fig. 80) attached to the opening of which is a strong rubber tube about three feet in length and filled with a solution of boric acid or sterile water. If the point of puncture is open to choice, about the seventh intercostal space is chosen in the middle or posterior axillary line, under all circumstances at a point where flatness and absence of fremitus are present.

Fig. 80.



Aspirator. According to Fürbringer.

If one is not entirely certain regarding the condition, an exploratory puncture should be done previously. The child is held firmly in the sitting position. Under antiseptic precautions the trocar is quickly introduced for about 2–3 cm. above the edge of a rib (the intercostal artery runs behind the lower border of the rib) and the stilet withdrawn. The effusion in recent cases is under absolute pressure and evacuates itself. A sudden interruption of the flow is caused by shreds of fibrin which are pushed back by the blunt needle. In large effusions the evacuation is prolonged by closure of the trocar from time to time, otherwise collapse may take place. Indeed, it is wise in cases of debilitated children or in large effusions to give coffee or brandy with water before the operation. As much of the fluid is evacuated as will flow out spontaneously. In long existing effusions very little or no fluid will flow out spontaneously because the pressure within the pleural cavity is not sufficient. In these cases the water filled Heber tube attached to the return opening of the trocar which after closure of the cock of the trocar is led vertically downward into a flask is of service. It itself aspirates slightly. If this is not successful, one of the well-known aspirators, Potain's, etc., is to be brought into service.

Instead, an apparatus, as pictured in Fig. 80 by which the aspiration is done with the mouth, may be inexpensively improvised, and will be just as good as those expensive instruments. If a simple trocar is possessed, the rubber tube, sterilized and filled with water, is pushed over to the single opening of the trocar and the aspirator is then attached. In case of necessity a large piston syringe will suffice. No force whatever should be used in aspirating. A large effusion must not be removed entirely, and at any rate one must desist as soon as violent coughing or weakness occurs or if the effusion becomes bloody. The puncture is closed with a collodion dressing. If paracentesis has not been done during the febrile stage the rest of the effusion usually becomes absorbed spontaneously, otherwise, and also during the existence of the original, additional punctures may become necessary.

Treatment of Empyema.—The sooner the pus is evacuated the more rapidly and easily will recovery take place. Early recognition will often prevent long illness and death. As soon as a pleurisy is recognized as being purulent no time must be lost with expectant treatment, but evacuation of the pus must proceed at once. A short delay is permissible only in cases of small empyemæ following pneumonia in which the exploratory puncture has yielded only a small amount of pus and the general condition is in a state of improvement, since pneumococcus empyemæ of small extent may often become absorbed spontaneously.

The following methods of evacuating the pus may be employed: simple puncture, Heber drainage, thoracotomy with or without resection of ribs. The point of selection is determined by the exploratory puncture (see above). If open to choice the median or posterior axillary line about the seventh or eighth rib is preferred, in thoracotomy the scapulary line.

1. The *simple puncture* as it has been described above should only be attempted in a recent pneumococcus empyema (metapneumonic empyema) of small area without large coagula, since in this instance recovery is exceptionally possible. The lumen of the trocar must measure at least 3 mm., and the pus is to be evacuated as completely as possible by aspiration. If recovery does not take place after two punctures at most, one must proceed differently. In strepto-, staphylo- and fetid empyema, puncture is to be abandoned from the outset. On the other hand, puncture (with Heber drainage) is the most appropriate procedure in tuberculous empyema, though even here resection of the ribs is frequently preferable even when a passably good pulmonary and general condition exists. Puncture is also often employed with advantage in ordinary empyema as a preparatory operation, whenever the patient comes under treatment in an urgent and very debilitated condition from an extensive effusion, and when one wishes to give him a few days' time to recover his strength for the operation to be undertaken.

2. The *Heber Drainage* often named after Bülau, but already used previously by Playfair and others, has been frequently employed during the last twenty years. After anæsthetizing the skin a trocar of at least the calibre of a lead pencil is introduced in the 6 – 7 intercostal space in the median axillary line. After removal of the stillet a rubber drainage tube as thick as possible is immediately passed through the cannula into the pleural cavity. The cannula is then carefully drawn over the tube, the end of which is immediately clamped so that no air can gain entrance. The tube is firmly fastened to the chest by a silk thread and collodion dressing. After removal of the clamp an additional rubber tube filled with boric acid solution is connected with the drain by means of an interposed glass tube, and is led into a glass vessel standing on the floor which is partially filled with boric acid solution (see Fig. 81). In this way the pus will be evacuated gradually into this vessel. The puncture wound in the chest wall enlarges spontaneously during the next few days, and the drainage tube is changed several times for still larger tubes. After about 14 days, simple drainage without aspiration tubing and a cotton dressing are employed (Orloff).

Fig. 81.

Heber drainage in empyema, according to Bülau.

The advantages of the Heber Drainage consist in this—that no anæsthetic is required and that the slight operation generally causes but little weakness. The advocates of this method see its greatest value in the prevention of a pneumothorax. The disadvantages are as follows: The flow of pus is often interrupted by large coagula which are frequently present in pneumococcus empyema. (The Heber Drainage, moreover, is especially recommended in streptococcic empyema which is comparatively rare in children.) The evacuation must then be accomplished by means of a piston syringe which is attached to the tube. Frequently, evacuation of the tough pus and shreds is very inadequate. The pus may re-accumulate, or the method may be a

failure from the first, so that thoracotomy must after all be undertaken subsequently.

The Bülau method does not give good results in small effusions, old empyema, and sunken thorax (with slight internal pressure). The after-treatment requires constant medical supervision and makes great demands on the attendants, so that the Heber Drainage is not well adapted to private practice.

3. *Simple thoracotomy* may be undertaken in younger children under a local anæsthetic of ethyl chloride; in older children slight chloroform anæsthesia is required. A longitudinal incision is made in about the 6–7 intercostal space closer to the lower than the upper rib, and after division of the intercostal muscles the pleura is freed in this situation, which usually does not demand the ligation of vessels. The pleura is then incised, the pus slowly evacuated (the wound being frequently plugged with the finger), and the opening enlarged to the size of the external wound by means of a blunt instrument. The large coagula are removed with the forceps. Irrigation of the pleural cavity is often harmful, and is attended by danger of collapse or convulsions, and is to be advocated only in fetid empyema. A thick, short drainage tube is placed in the wound. Dressing as in resection of the ribs is employed. Simple thoracotomy amply suffices in recent empyema (Foltanek), and is preferred by some to resection of the ribs (Koplik, Cautley, Blaker), especially in children under 18 months. Koplik recommends it in children over 18 months only when high fever, cardiac weakness, or pericarditis, are present. Should resection of the ribs become advisable later on, it can then be much better undertaken on the patient who has improved in strength.

Fig. 82.

Rib shears.

4. *Resection of the ribs* affords the surest method of thoroughly evacuating the pus. Under chloroform anæsthesia, an incision about 6–8 cm. in length is made directly to the middle of the rib (8 – 9 rib in the posterior axillary or scapulary line). The periosteum is elevated with the periosteum elevator, and about 5–6 cm. of the rib is cut away with the bone forceps (placed vertically from edge to edge, the periosteum elevator being interposed). A special rib-shears, such as has been in use for twenty-five years at Hagenbach's Clinic in Basle, is useful (see Fig. 82).

The ends of ribs not covered by periosteum and soft parts must not be allowed to remain. The pleura is then incised, the pus slowly evacuated, and the wound in the pleura enlarged according to the size of the piece of rib which has been removed. Schede recommends an additional transverse incision through the middle of the pleura, the intercostal

artery being tied, in order to be able to oversee the pleural cavity and to thoroughly remove large coagula with sponges. No irrigation. Schede places several very thick but very short drainage tubes in the wound, which reach merely to the pleural cavity, and are cut off at the level of the skin and fastened by means of large safety pins. A piece of protective silk the size of a hand and fitting snugly to the skin is placed over them. Over this a thick, aseptic dressing is applied, which may remain for several days if it does not become saturated. At any rate the second dressing should be allowed to remain for a long time, as this is important for healing. During the first dressing the lung often comes in contact with the chest wall once more (Schede), and expands again within a few days (Kissel). Therefore, the disadvantages of a pneumothorax, which the advocates of the Heber Drainage denounce, do not exist. The drainage tubes are soon removed with the exception of one, which is removed only when the discharge ceases and the vesicular breathing is heard next to the wound.

Rib resection may be undertaken by any physician, and is a much easier operation than for instance tracheotomy. It is an operation that brings extreme gratitude, since in favorable cases great relief is experienced, the fever disappears in from one to two days, and recovery rapidly takes place. As usual pneumococcic empyema furnishes the best prognosis, as it is cured in about six weeks. The prognosis depends materially upon the underlying disease. Of 288 collective cases 13.9 per cent. died of metapneumonic empyema, 32 per cent. of secondary and metastatic empyema. But even a pure pneumococcus pneumonia which often comes to operation in a hopeless condition may terminate fatally, from collapse or sepsis following the operation, or later from pulmonary abscess, sacculated portions of an empyema not having been touched by the operation, from purulent pericarditis, metastases, bronchopneumonia, etc.

Empyema necessitatis also requires operative interference; and likewise empyema rupturing through the bronchi, if recovery does not take place spontaneously. In bilateral empyema, one side is first operated upon, and the side which is only to be operated upon after some time is aspirated. It is of interest that bilateral resection of ribs undertaken simultaneously has been done without any bad results. Long-standing fistulous openings are especially observed in cases operated upon late, in which extensive adhesions and perhaps sacculated areas are present, or when an insufficient number of ribs have been resected, the pleural opening having been made too small.

In old empyema with tough cavity formation simple resection is frequently without result, but on the other hand Estlander's multiple resection of ribs or thoracic resection advised by Schede, *i. e.*, the removal of the ribs and the tough intercostal portions within reach

of the empyema cavity, are useful. Recently, in difficult cases even the stripping off of the thickened pleura from the pulmonary surface is advocated.

Reviewing the different methods of treatment of empyema, we cannot give preference to any one method. Thoracotomy, especially if combined with costal resection, always gives the most certain results. These measures should also always be used where the children are still strong and in older cases. Most surgeons, as well as many pediatrists (Hagenbach-Burkhardt, Baginsky), prefer costal resection from the first. In frail or debilitated children where it is desirable to omit anæsthesia the Heber Drainage may be tried in fresh cases; but one must always be prepared for failure. I myself have seen but little good result. In ichorous, in old empyema, in strepto- and staphylococcic empyema, in pyopneumothorax, it is to be discarded from the first. In a large number of cases under similar conditions, Schede found that costal resection yields far better results as regards mortality and recovery than the Heber Drainage.

The treatment of the remaining disturbances (thickening, contractions) following a successful operation for empyema is the same as for the sequelæ of serous pleurisy (see above).

Peripleuritis or phlegmon endothoracica is described as a rare abcess formation between the costal pleura and chest wall. The affection may occur primarily following injuries, or secondarily in perforating empyema, caries of the ribs, or actinomycosis. It causes circumscribed dulness and may rupture externally, causing numerous fistulæ. The difficult differentiation from a circumscribed empyema is made possible by the presence of the normal pulmonary sound below the dulness, and by good motility of the lungs during respiration.

Pneumothorax presents scarcely anything special in children, except in the manner of origin.

It is much rarer than in adults, and occurs more frequently independently of pulmonary tuberculosis, as in the course of bronchopneumonia and gangrene of the lungs, quite frequently in measles, next in pulmonary emphysema, diphtheria, whooping-cough, in foreign bodies in the lungs (Zuppinger), rupture of softened bronchial glands, and after injuries. I have seen a well-marked case without external injury, following a fall from a tree.

The **symptoms** and **treatment** are the same as in adults. In the presence of pleuritic adhesions only a partial pneumothorax is the result. At a later stage pyopneumothorax often develops. In a case where valvular pyopneumothorax and rapid general external emphysema of high grade developed after a subcutaneous fracture of rib in a four year old boy, threatened death was averted by thoracotomy (von Muralt).

CONGENITAL ANOMALIES, NEOPLASMS AND PARASITES OF THE LUNGS AND PLEURA

A congenital lack of development of a lung has been observed several times. The same is frequently recognized only in autopsy, since the affected side of the chest becomes expanded by vicarious emphysema, and since the development of the sound lung is retarded.

Pulmonary hernias are of rare occurrence, mostly congenital, more rarely following injuries. They are due to defects of the muscles or ribs in some part of the thorax, in which situation the lung protrudes externally in the shape of an elastic, compressible, and reducible swelling, increasing during expiration and producing clear pulmonary resonance. The costal pleura serves as a hernial sac; the overlying skin is normal in appearance. Retention by means of a bandage often acts beneficially.

Of *neoplasms* the sarcoma is the most frequently met with, and often occurs secondarily in the lungs, but only rarely primarily in the lungs or pleura. Secondary sarcoma (frequently multiple and central) often progresses without any symptoms. Primary sarcoma of rapid growth may lead to extreme flatness (also with pleuritic effusion), distention of veins in the chest, compression of the large venæ cavæ, and œdema of the face and the upper extremities, displacement of the heart, etc.

More frequently than primary, genuine neoplasms but with similar symptoms are seen *echinococci of the lungs*, in countries where this disease is also otherwise observed. Next in frequency to the liver, the echinococcus disease attacks the lungs (by direct immigration of the embryos or by metabolic invasion of a daughter-cyst), or the pleural cavity, by extension from the surface of the liver. The cysts are usually unilobular. The children are usually between six to fourteen years of age.

The **symptoms** are often for a long time those of an indefinite pulmonary or pleuritic affection. Unilateral dulness, most frequently in the region of the right lower lobe, dyspnœa, sometimes arching of the chest wall at a circumscribed spot, frequent attacks of coughing with mucopurulent, bloody expectoration in which remnants of hydatids or hooks are demonstrable, gradually develop. Intercurrent attacks of fever (pus formation of the cysts) often occur. The course is very slow, and often terminates fatally amid emaciation, bronchopneumonia, etc. Spontaneous recovery is sometimes possible by rupture through the bronchi.

Diagnosis is greatly facilitated by the presence of echinococcus of the liver; otherwise, simply a pleuritic effusion (which often accompanies it) or some pulmonary affection is assumed to be present. The diagnosis is rendered certain by the characteristic composition of the expectoration, or by the contents of an exploratory puncture (clear, without albumin).

The **treatment** should not cause suppression of the cough (Roger), since this facilitates the expectoration of the membranes. Opening of the cyst by costal resection may often still lead to recovery.

DISEASES OF THE LARYNX

BY
Dr. DEMETRIO GALATTI, of Vienna

TRANSLATED BY
Dr. CLEMENT F. THEISEN, Albany, N. Y.

ANATOMY AND PHYSIOLOGY OF THE CHILD'S LARYNX

The larynx of the child differs from that of the adult. The two plates of the thyroid cartilage are not united at such a sharp angle as in the adult, and their section forms an almost semicircular arch. The line of union in the middle can hardly be felt. A further difference is the tendency for the cricoid plate to incline posteriorly. This is most marked in the newborn infant, and disappears about the fourth year. The nicking of the laryngotracheal tube during the first year of life is explained by this anatomical fact. The glottis of the nursing infant is proportionately much smaller than in the adult, but this peculiarity of the child's larynx gradually disappears as further development takes place. It is believed that the larynx does not develop much between the fifth or sixth years and the age of puberty. After this period is reached however, the larynx grows rapidly. In the male, there is a change in the shape of the thyroid plates, and the laryngeal prominences are formed. Figs. 83 and 84 represent the relation between the fœtal and the adult larynx (vertical sections).

In Figs. 83 and 84, the nicking of the laryngotracheal tube, and the separation of the thyroid cartilage from the cricoid cartilage, which, in relation to the glottis, is much greater than in the adult, is clearly represented. The thyrohyoid region is worthy of note. The hyoid bone rests on the upper edge of the thyroid cartilage. For this reason, the thyrohyoid space is much lower than in the adult. A distinct median and lateral thyrohyoid ligament, only appears in the seventh year.

The physiology of the child's larynx has been only slightly investigated. The physiological relations of the movements of the separate laryngeal cartilages to each other have not been studied at all. The peculiarities of the child's larynx are associated with it's anatomy, and the changes which develop as the child grows older, play an important rôle in voice production. This however has no important bearing upon the pathology of the child's larynx.

SOME GENERAL REMARKS CONCERNING THE EXAMINATION AND TREATMENT OF THE LARYNGEAL DISEASES OF CHILDHOOD

Laryngoscopy, the universal method of examining the adult larynx, cannot be frequently employed in the laryngeal diseases of childhood.

FIG. 84.

FIG. 83.

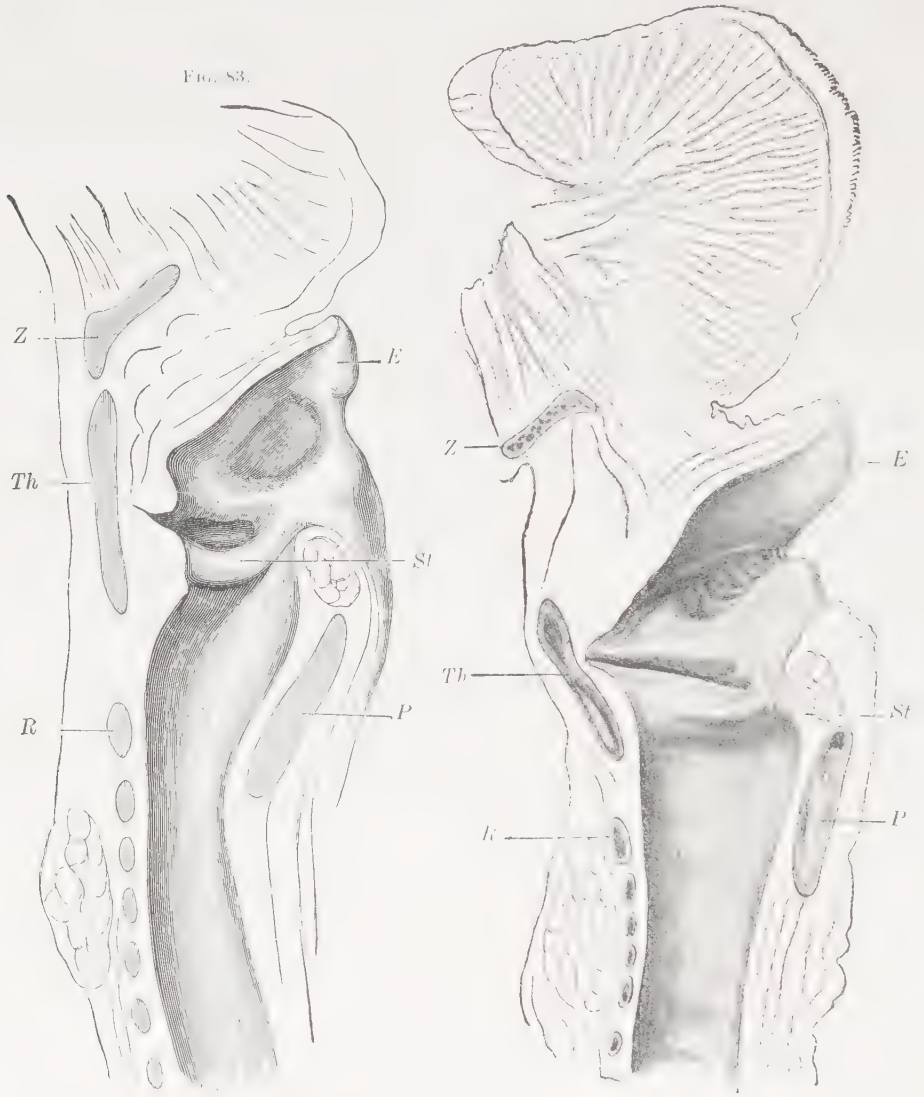


Fig. 83 represents the larynx of the newborn infant enlarged to the dimensions of the adult (male) larynx shown in Fig. 84. Fig. 84 shows a sagittal section of the adult male larynx. *Z.*—Hyoid bone; *E.*—Epiglottis; *Th.*—Thyroid cartilage; *St.*—True vocal cord; *R.*—Cricoid ring; *P.*—Cricoid plate.

This is so, partly because of the type of disease, and partly because of the patient to be examined. The symptoms are often so alarming, that the physician must give relief without much of an examination. The symptoms are usually those caused by stenosis, and attempts

to examine the larynx will only make them worse. Stenoses of the larynx play a very important rôle in childhood. Slowly developing diseases of the larynx, permitting a laryngeal examination are fairly rare in children.

It is most difficult to make a laryngeal examination in children between the fourth and tenth years. The child struggles, and shuts the teeth tightly, making the use of an assistant and a mouth gag necessary. After the child is wrapped in a blanket it should be firmly held in the upright position in the lap of an assistant, the head being pressed back against the assistant's chest. Even then, the child's cries and the excessive mucus secretions in the throat, will often make the examination impossible. In older children, the examination is more easily accomplished. Forcible examination should not be attempted. The larynx may be often inspected by Kirstein's autoscopy. If the tongue is depressed in front, the epiglottis may be seen easily, and it is not necessary to carry the head of the child well forward as in the case of adults. An inspection of the epiglottis is valuable, because many diseases are located in this region. An examination with the finger, and external palpation should not be neglected. In cases of foreign body, the X-ray examination will be valuable, if the child does not struggle too much.

Symptomatology.—This is similar to that in adults, except that the symptoms are usually more severe, and may rapidly become dangerous. When inhalation methods of treatment are employed, the air of the room should be saturated with the remedies. For this purpose either steam alone, or combined with oil of eucalyptus, oil of turpentine or menthol, may be employed.

FOREIGN BODIES IN THE LARYNX

These are very common during childhood. In rare cases, they may come from within (inhaled masses of mucus, or ascarides). Anything that the child plays with, if small enough, may be inhaled into the larynx, such as buttons, needles, seeds, bullets, or particles of bone or food. Food particles may be inhaled if the child is suddenly alarmed while eating and takes a deep inspiration. Small bodies inserted in the nose may also be inhaled into the larynx. Pieces of instruments have entered the larynx during operations on the tonsils. The situation of the foreign body is usually either above or under the glottis. Sharp bodies may penetrate the tissues to such an extent that they are not easily seen or felt.

The **symptoms** will depend to a certain extent upon the size and shape of the foreign body. In adults, it may remain in the larynx for a period without causing any symptoms, but in children, dangerous symptoms usually develop, even when comparatively small bodies are inhaled.

If the foreign body is not large, the first symptoms may be severe coughing. These paroxsyms of coughing often expel the foreign body. If is it not soon coughed out, a bloody secretion is thrown out, particularly in cases of pointed foreign bodies. Pain and spasm of the glottis develop, the laryngeal mucosa becomes hyperæmic, and the tissues around the foreign body become swollen. Hoarseness is also present. A purulent expectoration follows the increased inflammation. Severe inflammatory processes, with serous or phlegmonous œdema, abscess, perichondritis, or erosions of blood vessels may also occur. If healing takes place either spontaneously or after some operative procedure, symptoms of stenosis due to the presence of cicatrices often remain. A forcible examination of the larynx is particulary dangerous in this class of cases. Sometimes the larynx may be inspected after the use of strong cocaine solutions (20 per cent.), but in little children, retching and excessive mucus secretions are produced, and a general anæsthetic for the purpose of performing laryngoscopy, and at the same time removing the foreign body, will have to be employed. Eucain solutions are not as good as cocaine solutions. If suffocation is not imminent, with patience a laryngeal examination may finally be made. The foreign body may sometimes be felt with the finger. A digital examination is however not without danger.

If the **diagnosis** of a foreign body cannot be made with certainty, the possibility of a lateral pharyngitis, which also produces the feeling of a foreign body in the throat, must be considered. The diagnosis of this condition may be made by direct inspection and by the course of the disease.

Croup must also be differentiated from this condition. In doubtful cases, the long-continued coughing and stenosis, and the sudden onset in a healthy child will clear up the diagnosis.

Prognosis.—This is often unfavorable, because in cases where large foreign bodies are present, suffocation is imminent, and the tardy removal of smaller bodies will be followed by serious symptoms.

Treatment.—Foreign bodies that are not imbedded, may be expelled during coughing. Even when imbedded, the resulting suppurative process may loosen them so that they are thrown out. The use of emetics is not without danger, because a deep inspiration may carry the foreign body further down. This is also a danger during attempts to remove the body, and it should not be extracted until it is firmly grasped in the blades of the forceps. Some form of laryngeal forceps is best for this purpose.

In children however, when the larynx cannot be directly inspected, this method of removing foreign bodies is not feasible. Tracheotomy and the removal through the wound is best. When pieces of metal have been inhaled, a magnet may be used through the wound.

When there is danger of suffocation, tracheotomy, even in cases of larger foreign bodies, is indicated before attempts to locate the body are made. In cases of impacted foreign bodies, the wound may have to be enlarged upwards, care being taken to protect the vocal cords. After removal, the hyperæmia may be relieved by ice compresses and ice in the mouth.

Adrenalin may have a valuable place in this respect in the future.

BURNS OF THE LARYNX

Of the many forms of injuries to the larynx in childhood, burns are most frequent. They are mainly accidental, due to swallowing lye or boiling water. Some of the lye may reach the interior of the larynx, causing injuries there as well as to the epiglottis. Tissue sloughing, pain in swallowing, hoarseness, œdema, perichondritis, and ulcers, leading to stenosis, may result.

The diagnosis is made by the presence of burns on the lips, and in the mouth and pharynx, so that this condition is not easily mistaken for diphtheria.

In cases of lye poisoning, antidotes such as acids (vinegar and water), should be employed. Ice may be melted in the mouth and ice compresses used.

PAPILLOMA OF THE LARYNX

Malignant neoplasms in the larynx during childhood, when they occur, are usually in the form of the sarcoma or epithelioma. Benign growths are much commoner, particularly three varieties; the singer's nodes, the granuloma, and the papilloma. The nodes usually result from a catarrhal laryngitis. The granuloma accompanies operative procedures, and the pressure ulceration following intubation.

The laryngeal papilloma occurs more frequently in boys than in girls. It is sometimes congenital, and may occur as a result of inflammatory affections of the larynx. It may be single or multiple, and sometimes attains a large size. It is usually situated on the true vocal cords, and either takes the form of a simple smooth node, or a lobular growth as large as a raspberry, with a broad base. Small papillomata do not always produce disturbances. But as a rule new growths in the larynges of children cause more trouble than in adults.

The first **symptom** is a change in the voice, which becomes worse until complete hoarseness results. Cough is usually present, and as the growth develops, difficulty in breathing, to the point of alarming suffocative attacks, occurs. In some of the cases the symptoms develop slowly. This is particularly true of the congenital cases, in which the papilloma grows very slowly.

In another class of cases, the course is rapid, necessitating prompt

attention. In still another class, the symptoms gradually subside, and finally disappear entirely. In such cases the growth disappeared spontaneously.

The **diagnosis**, and there is usually time enough to make it, is settled by a laryngeal examination.

The **prognosis** is favorable, first because of the chance for a spontaneous cure, secondly because this form of new growth does not develop into carcinoma, and thirdly because after removal recurrences are not frequent.

Treatment of the papilloma itself is not always necessary. Papillomata have been coughed out spontaneously. Operative interference is usually necessary however. Intubation has been successful in some cases, but the best method of treatment consists in the endolaryngeal removal of the growths, many sittings being often necessary. Laryngo-fissure must often be employed, and when urgent symptoms arise, tracheotomy should be performed.

ACUTE CATARRHAL LARYNGITIS

Acute catarrh of the larynx is more common in children than in adults, because the laryngeal mucous membrane is more sensitive in children, and catarrhal laryngitis is also a symptom of certain infectious diseases. It occurs more frequently in boys than in girls, and particularly in anæmic, rachitic, and scrofulous children. These conditions seem to bring about almost a predisposition to catarrhal laryngitis. Primary catarrh of the larynx, may be caused by exposure to cold, the secondary catarrhal conditions, complicating the infectious diseases, particularly measles, influenza, and whooping-cough. Sudden changes in the temperature with strong east and north winds, in spring and fall, are also etiological factors.

Bacteriological examinations in cases of laryngitis are usually negative, but varieties of pneumococcic and streptococcic laryngitis have been described. B. Fränkel has described a peculiar form of laryngitis, occurring with influenza, which is characterized by the presence of small white points.

Ordinarily, only the mucous membrane is involved in the inflammatory process, and this varies from a mild to the most severe inflammation. In some cases the membrane is only slightly reddened and not swollen, while in others the mucosa is dark red and much swollen. There is an increased mucus secretion which in the mild cases is white; in the more severe cases it dries and clings to the mucous membrane in the form of crusts (laryngitis sicca).

Symptoms.—In the severe forms of catarrhal laryngitis, the secretion becomes mucopurulent, and there may be superficial erosions of the epithelium. There is a form of laryngitis involving only the portion

of the larynx below the vocal cords (subcordal or hypoglottic laryngitis), in which the symptoms may be very severe. In this form, recurrences are common, and predisposition plays an important rôle. It is often the result of a tracheitis, and then the vocal cords may be normal. There is great redness under the cords however, the swelling being often slight, but at times being so considerable that respiration may be embarrassed. The typical symptom-complex which has given this affection the name "false croup" is present when this condition develops. A primary laryngéal catarrh may begin with some temperature elevation, and it is very often preceded by a coryza lasting for several days. Then with the inflammation of the pharynx and larynx, cough, some pain in the throat, disturbances in deglutition and changes in the voice develop.

The fever runs its course in from one to three days, the cough which is at first dry becomes looser with expectoration, and in about a week the attack subsides. Cases of pseudocroup run an altogether different course. The child, who may have had a slight coryza, wakes up during the night with alarming symptoms.

Respiration is audible and difficult, there is the characteristic barking cough with some cyanosis of the face. Hoarseness to the point of aphonia is present. The attack lasts from a few minutes to several hours, and rarely recurs during the night.

In the mild cases, the child soon goes back to sleep and dyspnœa subsides. The voice becomes clearer and there is a mucus expectoration.

In the severe attacks, alarming suffocative symptoms come on, and death has occurred.

On laryngeal examination, the mucosa may not appear sufficiently swollen to account for the symptoms, and many authors attribute these attacks to a spasm of the glottis brought on by the laryngeal catarrh. The child breathes with difficulty and there is cyanosis of the face. Such attacks may last for hours. As soon as the attack begins to subside, the child perspires and nothing remains but the cough and hoarseness. As a rule, no attacks develop during the succeeding night, except in some rachitic children. Fever may be present with the onset of the attack, and may last for a few days. At times the dyspnœa is slight while the cough is very severe. In uncomplicated cases, the attack runs its course in from five to ten days. Bronchial catarrhs and even pneumonia are not uncommon complications of such attacks.

A chronic catarrh sometimes follows the acute process. In some cases, particularly in false croup, the mucus becomes dry and thick, causing the dyspnœa, which ceases as soon as the crusts are expelled.

The **diagnosis** of the mild form of catarrhal laryngitis is easily made, but in cases of pseudocroup, true croup must be thought of. In pseudocroup, the membranes on the gums, tonsils, pharynx and

larynx are absent, and the sudden onset at night, with the decided relief in a few hours is characteristic.

Prognosis.—The prognosis of simple catarrhal laryngitis is favorable. False croup is more serious because of the possibility of suffocation, and complications like bronchitis and pneumonia. These complications are more apt to develop when influenza or measles have caused the laryngitis.

Treatment.—The child should be kept in a warm moist temperature, the air of the room being saturated with steam. Drugs are unnecessary in mild cases, and no attention need be paid to the temperature.

For the dryness of the throat, warm drinks (warm lemonade, tea or milk), may be given to favor free perspiration. When this begins, the laryngeal secretion will also start. Warm, moist compresses to the neck are also useful. After the first day, the feeling of dryness in the throat can be relieved by inhalations of water or saline solution. When the cough is severe, with little expectoration, heroin. mur. 0.02 Gm.: 100 c.c. ($\frac{1}{3}$ gr. to $3\frac{1}{2}$ oz.), small teaspoonful every 2 or 3 hours, or morph. mur. in the same doses, may be given, or apomorph. mur. 0.03 Gm. ($\frac{1}{2}$ gr.), acid mur. dil. 0.5 Gm. (ɱ 7), aq. dest. 150.0 c.c. (5 oz.), teaspoonful every 3 hours). As soon as there is free secretion, expectorants like ipecac should be used.

In severe forms, when the symptoms depend upon excessive secretion of mucus, emetics should be given, the best being tartar emetic, one dessertspoonful of a 0.2 per cent. solution, which may be repeated in fifteen minutes. In cases of pseudocroup, foot baths, with ice in the mouth and cold compresses to the neck, with steam inhalations and expectorants are useful. Tincture of aconite and belladonna, two or three drops every two hours will sometimes do good. When the difficulty in breathing becomes alarming, either intubation or tracheotomy must be resorted to. If an intubation tube can be left in long enough this method is often successful. If not tracheotomy must be performed.

Prophylaxis.—It is the duty of the physician to prevent recurrences if possible. To accomplish this, the powers of resistance of the individual should be increased. Susceptible children should not be permitted to go out in bad weather and extreme temperature changes should be avoided. The child may be made less susceptible by proper attention to clothing, gymnastic exercise, fresh air, and ventilation of rooms. Most important of all is a cold sponge bath at least once a day. Swimming is very good. When possible a change of climate is often effective.

CHRONIC CATARRHAL LARYNGITIS.

Chronic laryngitis is sometimes the result of a badly treated acute laryngitis, but it is mainly caused by voice strain, chronic catarrh of the nose and pharynx or adenoids.

It is comparatively rare during childhood. It localizes itself in the same way as the acute catarrh, so that partial catarrhal conditions, (chronic hypoglottic laryngitis), may occur.

The laryngeal examination shows a slight redness and thickening of the affected parts. Singer's nodes and ulcerations are rare in children. The inflamed parts are covered with either a thin, purulent, or dry secretion.

Symptoms.—There is a feeling of dryness, with enough irritation to produce some coughing and scraping of the throat. The voice is either husky or hoarse. Dyspnœa is uncommon, but cases occur with alarming suffocative symptoms.

Chronic hypoglottic laryngitis is characterized by attacks of dry, metallic cough, occurring at night, at intervals of several weeks. Some dyspnœa is present.

The **diagnosis** must be made with the laryngeal mirror. This condition must be differentiated from tuberculosis (rare in children), and from the symptoms caused by enlarged glands.

Prognosis.—A complete cure is not always obtained. Mild cases are often cured simply by the continued use of saline or charged mineral waters. As a rule, inhalations or insufflations must be employed. As inhalations, astringents, like tannin, or solutions of salt, or carbonate of soda may be used. The constitutional treatment is the same as for acute catarrh. Any nasal or pharyngeal catarrh, or adenoids, should be properly looked after.

SEROUS AND PHLEGMONOUS ŒDEMA OF THE GLOTTIS

By œdema of the glottis is meant an œdematous swelling of the submucous cellular tissue of the larynx, and the intermuscular cellular tissue of the vocal cords. This may be of a serous or inflammatory nature.

Serous infiltration of the submucous cellular tissue, occurs mainly as a complication of certain general diseases, such as chronic nephritis. It may be the first symptom of this condition. It occurs also with acute nephritis and heart troubles. It is also produced by stasis, through tumors compressing veins in the neck. The œdema caused by the administration of iodide of potash should also be considered. Alarming œdema has occurred after small doses of iodide.

The *inflammatory œdema* is more common than the serous variety. Part of the cases belonging in this class, have been described under the name submucous phlegmonous laryngitis. Inflammatory œdema, is the result of inflammatory, or ulcerative local processes, of not only the larynx, but the surrounding tissues as well. It occurs most frequently after laryngeal injuries (foreign bodies and burns), or with ulcers of the larynx. It also complicates perichondritis, erysipelas, phlegmon of the neck, and infectious diseases.

The usual *location* of the disease is in the aryepiglottic folds and epiglottis, more rarely in the vocal cords, interarytenoid fold and petiolus. The œdema complicating constitutional conditions, is more apt to be symmetrical than the œdema due to local conditions.

Symptoms.—In œdema of the epiglottis, the epiglottis and the connective tissue around the base of the tongue, are swollen. The examining finger feels the œdematous epiglottis, and a swelling on either side of it. The condition may be directly inspected if the tongue is pulled forward. The mucous membrane is reddened. Serum is not always evacuated when an incision is made. Pain in swallowing may be present with this variety of œdema, but rarely difficulty in breathing or hoarseness. Œdema of the aryepiglottic folds, is most important, because it may assume such proportions that the laryngeal lumen will be seriously occluded, causing alarming dyspnœa or suffocation. Œdema in other parts of the larynx is not so apt to embarrass respiration.

Diagnosis.—This can be made with the finger or by laryngoscopy. The serous or inflammatory nature of the diseased parts may be recognized by their color. The heart and urine should be examined.

A beginning phlegmonous œdema, cannot be differentiated from a severe laryngitis by laryngoscopy, but by the history, and by its course. It is sometimes impossible to differentiate a phlegmonous œdema from perichondritis.

The **prognosis** depends upon the cause and severity of the œdema.

Treatment.—This will depend upon the cause of the condition. When due to iodide of potash its administration should be stopped at once. In cases of nephritis, injections of pilocarpine ($\frac{1}{4}$ or $\frac{1}{2}$ syringefull or a 1 per cent. solution) are best, and will sometimes cause the œdema to disappear in a short time. Heart troubles should be treated.

Local Treatment.—When there is no danger of suffocation, ice in the mouth and cold compresses will be useful. The author has had no experience with adrenalin applications. When dyspnœa is great, scarification may be resorted to, but it will rarely be found effective. When pus is suspected, a deep incision is better than scarification. When difficulty in respiration is very great, intubation may be tried first, followed if necessary, by tracheotomy. The œdema is not relieved by intubation, and it only serves until tracheotomy can be performed. After tracheotomy the œdema may be treated locally.

LARYNGEAL PERICHONDRITIS

Idiopathic, as well as secondary perichondritis, is rare in childhood, the former, because it is an unusual condition anyway, and the latter, because its main cause, tuberculosis and syphilis of the larynx, are rare during childhood.

Perichondritis occurs most frequently with ulcerations in the larynx complicating infectious diseases, such as typhoid fever, varicella, measles, and scarlet fever, septic diphtheria, syphilis, tuberculosis, or as the result of a metastatic deposit.

Symptoms.—The arytenoid and cricoid cartilages, are most frequently involved, more rarely the epiglottis and thyroid. It is not limited to one cartilage. It is accompanied by high temperature, which may be modified by the fever going with the underlying condition. A metastatic perichondritis, begins with chills and pain in the larynx. The diseased cartilages cause pain during the entire course of the disease, and are sensitive to pressure.

Difficulty in swallowing is always present, being most marked when the epiglottis, the arytenoid and the thyroid plate are involved. Hoarseness and difficulty in breathing accompany the swelling of the perichondrium. The formation of an abscess may entirely occlude the lumen of the larynx, so that the dyspnœa will persist until the abscess opens spontaneously or is incised.

The difficulty in breathing is least with involvement of the thyroid cartilage with external abscess formation. In such cases the subcutaneous abscess can be seen and palpated. The dyspnœa is not always entirely relieved when the abscess is opened, because the necrosed cartilage may act as a foreign body in the larynx. A laryngeal examination, which at the same time clears up the diagnosis can often be made. When the abscess is opened and the cartilage thrown out, the ulceration heals with the formation of cicatricial tissue, causing deformity and stenosis of the larynx.

The **course** of a perichondritis is rapid in septic and acute purulent processes, and usually slow in cases of syphilis and tuberculosis.

Diagnosis.—The diagnosis is not always easy even when it is possible to make a laryngeal examination, it is difficult in the beginning to make a differential diagnosis between perichondritis and a phlegmonous or other severe inflammatory affection.

The history of the case is not always decisive. In ulcerative conditions, either phlegmon or perichondritis may result. The course of the disease is quite characteristic. When an external abscess has formed, it must be differentiated from a glandular inflammation or cyst of the thyroid gland. The symptom-complex will decide this.

The **prognosis** depends upon the underlying disease, but is serious in every case, because there may be danger to life. The pronosis is most favorable in syphilitic cases. A chronic hoarseness and stenosis must always be considered.

Treatment.—The underlying disease must first be conquered, the syphilitic cases being the most favorable for treatment. Local treatment consists in the use of leeches or ice compresses, and ice in the

mouth for the pain in swallowing. The abscess may be opened endolaryngeally, or, when subcutaneous, from without. The sequestrum must also be removed, and finally the resulting stenosis treated, in the accepted way.

STENOSIS OF THE LARYNX

Stenoses of the larynx are extra- and intralaryngeal in nature. The former are also known as compression stenoses; they are produced by compression of the larynx by a goitre, lymphatic glands, abscesses, etc. Syphilis, laryngospasm, cicatrices following operations, more rarely tuberculosis, and congenital malformations, may also cause stenosis.

Symptoms.—A prolonged audible inspiration is one of the main symptoms of laryngeal stenosis. The thorax becomes widened, and there is a drawing in of the epigastrium and the intercostal spaces. During inspiration, the larynx sinks, the accessory muscles of respiration (the sternocleidomastoidei, the omohyoidei, the pectorales, serrati and rhomboidei), are brought into play, the alæ nasi are dilated, and the face becomes pale. Later respiration is increased, the face is cyanotic, and there is a cold sweat.

The attack may finally end in death by suffocation. There is a marked inspiratory stridor, while expiration, during which the larynx rises again, is easy and noiseless.

Laryngeal stenosis differs from tracheal stenosis by the movement of the larynx and the inspiratory stridor, which may be palpated, and by the bending backwards of the head.

Expiratory stridor is rare in laryngeal stenosis, while it is characteristic of tracheal stenosis. The head is bent forward in tracheal stenosis. The severity of the symptoms depends upon the extent and situation of the stenosis, and the rapidity of its development; the more rapidly the stenosis develops, the more severe are the symptoms.

What aids to **diagnosis** has the physician who is called to attend such a case, and cannot examine the child's larynx?

The **history** of the case is important although not always exact. If a positive history of a foreign body or burn is obtained, the diagnosis is evident. When the history of swallowing a corrosive substance cannot be obtained, then previous illnesses, and the rapidity of the onset of the stenosis, are important. The onset is rapid in cases of phlegmon of the neck, inflamed glands, hæmorrhages in thyroid cysts, retropharyngeal abscesses, large foreign bodies in the œsophagus, foreign bodies in the larynx, burns in the larynx, pseudocroup, œdema, diphtheria, perichondritis, ulcers, in acute infectious diseases, and spasm of the glottis. It is slow in tumors (including goitre), congenital malformations, new growths, chronic ulceration (tuberculosis,

syphilis), cicatrices following chronic ulcerations, after perichondritis, and after intubation and tracheotomy.

The further history as to whether the stenosis developed suddenly or only rapidly is of importance.

Sudden stenoses point to foreign bodies, burns, pseudocroup, and spasms of the glottis, while those developing less suddenly point to œdema of the larynx and diphtheria.

A sudden occlusion of the glottis may also be caused by pedunculated new growths.

It must also be determined whether there have been any previous illnesses, such as measles, scarlet fever, typhoid fever, whooping-cough, and other acute infectious diseases, which cause false croup, ulcers, perichondritis, nephritis or œdema of the glottis, or whether the child has rachitis with which spasm of the glottis occurs. If there have been previous attacks, the diagnosis of false croup is probable. Congenital hoarseness points to congenital malformations, papillomata, or syphilis. An eruption coming on soon after birth also suggests syphilis. The neck should be examined for glands, goitre, tumors, phlegmon, or for an abscess connected with the laryngeal cartilages. In such cases, a diagnosis of compression stenosis or perichondritis can be made; it must be remembered however, that an endolaryngeal stenosis may exist in conjunction with the goitre. If fever, which cannot be traced to any other disease is present, it is suspicious of either diphtheria, acute catarrh with pseudocroup, abscess or perichondritis. The latter conditions would be the more probable if pressure against the larynx produces pain.

An examination of the heart, urine, and osseous system should also be made. By this the possibility of œdema of the glottis or spasm of the glottis may be determined. A long-continued discharge from the nose is rather suggestive of a chronic laryngitis. A discharge that has only been going on for a short time, points to acute laryngitis or diphtheria.

Skin eruptions are valuable aids to the diagnosis particularly in suspected syphilis. Finally the mouth should be examined. Burns about the lips, the mucosa of the mouth or tongue, point to a similar state of affairs in the larynx. Diphtheritic deposits on the tonsils, pillars of the fauces, uvula, or pharynx, will of course decide the diagnosis of the laryngeal condition. The same is true of syphilitic ulceration.

The digital examination will decide the presence of an œdema of the larynx or retropharyngeal abscess. A direct inspection of a portion of the larynx is most useful.

The description of the methods of examination in cases of laryngeal stenoses, has, at the same time, pointed out the importance of the symptoms in arriving at a diagnosis.

The last resorts in the treatment of stenoses are intubation and tracheotomy.

DISEASES OF THE THYMUS, STATUS LYMPHATICUS AND SUDDEN DEATH IN INFANCY

BY

Dr J. K. FRIEDJUNG, of Vienna

TRANSLATED BY

Dr. WM. A. NORTHRIDGE, Brooklyn, N. Y.

The thymus gland is of considerable size in infants and is found in the upper anterior mediastinum. It is made up chiefly of reticular tissue. It begins to take its part in the making of the blood during the last fœtal months; and grows according to Waldeyer, until the child is one or even two years of age. After this it remains stationary until after puberty, when it gradually diminishes in size, undergoing fatty degeneration.

As found post mortem, the size and weight of this gland-like organ varies considerably. Friedleben has established the following average figures, and they have been frequently confirmed.

Weight of gland at birth	14.3 Gm.	214.5 gr.
From one to nine months	20.7 Gm.	310.5 gr.
From nine to twenty-four months	27.3 Gm.	409.5 gr.
From two to fourteen years	27.0 Gm.	405.0 gr.
From fifteen to twenty-five years	22.1 Gm.	331.5 gr.
From twenty-five to thirty-five years	3.1 Gm.	46.5 gr.

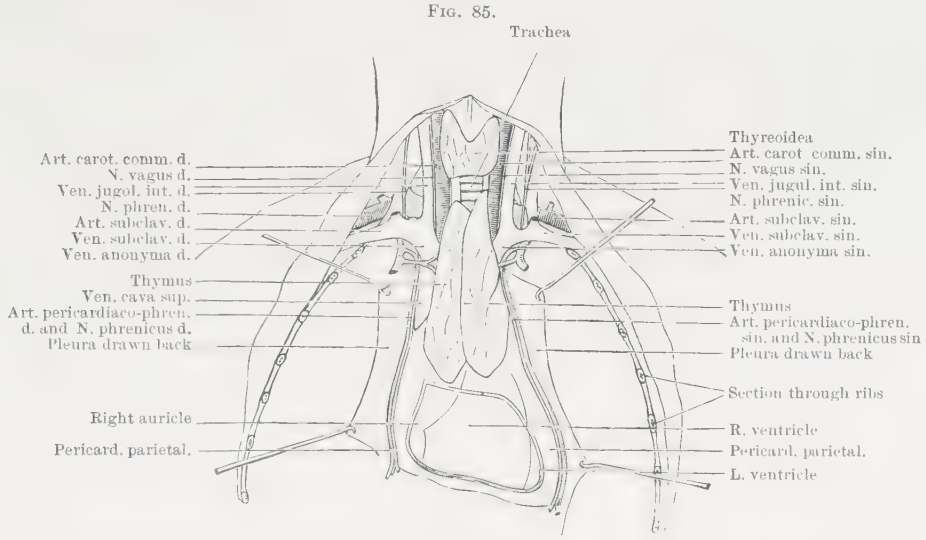
Waldeyer has found the remains of the thymus even later in life.

Anatomy.—The thymus consists of two lobes, faintly red in color. They are more or less pointed towards the upper part and rounded off toward the lower. They are bound together by loose connective tissue. The greater portion of the gland lies behind the manubrium and body of the sternum; but the sides and lower portion are covered by the folds of the mediastinum and are forced away from the chest wall by the anterior borders of the lungs. This location explains the peculiar normal percussion note of the thymus which is alluded to later on. It covers the pericardium and the beginning of the great vessels posteriorly and also reaches down to the pulmonary veins. The upper pointed edges of both lobes cover the trachea. On the sides, the thymus is bounded by the innominate and common carotid arteries, the vagi and the phrenic nerves. Back of the lobes, and in front of the vertebral column, are found the sympathetic nerves. The neighborhood of so many important vital organs (see Fig. 85) renders them liable to serious injury in case of disease with enlargement of the thymus.

Researches as to the *functions of this organ* are not yet concluded. While formerly the thymus was thought of only in connection with the formation of the blood, there are now several authorities who ascribe to the gland a secretion like other glands. It is also supposed to regulate the nutrition and growth of the bones and the brain, and also to act upon the circulatory system, raising and lowering the blood pressure.

In experiments upon animals, it has been observed that intravenous injections of the thymus extract are often fatal, the animal dying in convulsions (Abelous and Billard, Svehla, Basch).

FIG. 85.



Position of the thymus. The preparation illustrated above is from a child two and one-half years of age. The arteries were injected. The sternum and the greater part of the ribs were removed, and the lungs were cut away at the hilus. The mediastinal membranes were separated from the posterior side of the sternum, were loosened from the thymus and the pericardium, and fixed on the sides with hooks. The phrenic arteries and phrenic nerves are exposed. The posterior portion of the parietal membrane of the pericardium is removed, thus making the front portion of the right ventricle visible.

Observers are far from agreeing as to the consequences of extirpation of the thymus in young animals, for instance, Basch claims to have noticed a considerable increase in the elimination of lime, and considerable interference with the growth of the bones, after its removal; on the other hand, Sinnhuber and lately Fischl insist that there are no characteristic consequences following the operation.

Pathology.—Since Friedleben, by his thorough researches, reduced to a minimum the importance of the thymus in the diseases of infancy, many other voices have been raised in doubt during the last twenty years; so that at the present time we are in the thick of contradictory opinions.

I. *Hyperplasia of the Thymus.*—The greater portion of these discussions is taken up with arguments as to whether hyperplasia of the

thymus does or does not exist. The question is not an easy one to answer—When the clinician and the anatomist cannot agree who shall guide the pathologist?

The weights mentioned above are average figures, and are of no great value because the actual weights vary to so great an extent. For instance, at the end of the first month of life it varies from 2 to 31 Gm. (30–465 grains). In consequence, the statements of authors in regard to hyperplasia of the thymus, end generally as physiological studies. Occasionally one finds here and there extraordinary figures; as for instance, Köppe's case weighing 52.9 Gm. (793.5 grains). Again, Richter calls attention to the fact that thymus glands of considerable weight are quite frequently found. If one takes the dimensions of the organ as a guide, he should remember that differences in the histological conduct of the same sized glands, may produce the most different weights.

Fig. 86.

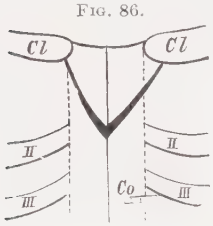


Chart showing average percussion dulness of the thymus in 22 cases from one month to one year of age. *Cl.*—clavicle, *II, III.*—2nd and 3rd ribs, *Co.*—upper border of heart dulness.

The anatomist is also always in danger of erring, as the thymus, keeping step with the general nutrition, will be found small in cases where wasting diseases have been present, and thus the size be underestimated, while on the other hand, he will overvalue the large thymi which he finds in well nourished individuals who have died after short illnesses.

The skeptical anatomist will in consequence not acknowledge that hyperplasia is possible; or if he does will claim that it can occur only in exceptional cases. Even the compression of the trachea, which has for long been considered a sure proof, has now lost credit, since Richter produced it artificially, showing instructive preparations.

The question for the clinician is not any easier. Palpation of the thymus does not give any points by which to estimate its size. The deformity known as pigeon breast caused by thorax-rachitis, is not even remotely caused by an enlarged thymus.

Percussion of the thymus has been very carefully studied out by Blumenreich, and is of much greater value than palpation. The dulness (diminished resonance) of the thymus covers a space the shape of a somewhat unequal triangle whose base is formed by a line drawn between the two sternoclavicular joints, and whose rounded off point lies downward on about a line with the second rib, or a little lower, and whose sides reach asymmetrically up across the sternal lines; on the left side more so than on the right. Between the thymus dulness and the heart dulness there is a zone where in normal cases the normal percussion note over healthy lung tissue is heard, because of the situation of the borders of the lungs. If this zone be dulled, or if these borders be lapped over one cm., then one may diagnose a very large thymus,

if all other causes of dulness have been excluded. This dulness on percussion can only be found up to the sixth year of life. Afterwards it lessens more and more.

Enlarged and caseated mediastinal glands are often the cause of mistaken diagnosis. These are found particularly in scrofulotuberculous infants. According to my experience, when other glands in this region are enlarged the same mistake is liable to occur.

When one can establish without a doubt, that the area of thymus dulness is greater than normal in any given case, without other disease being present it may then be stated that a diseased enlargement of the thymus is present.

The same rule applies to the results established by Hochsinger, by the use of the *Röntgen rays:* when showing the enlarged thymus. The middle shadow thrown by the thymus under normal conditions, rests on the shadow thrown by the heart, just as the slender neck of a bottle is fixed on the body of the bottle. In many cases this shadow of the thymus is more or less spread out. In these, after careful clinical examination, enlargement of the thymus was suspected. But sometimes one receives such pictures when all the clinical symptoms of an enlarged thymus are absent.

Are these cases of hyperplasia? Aside from quite isolated and monstrous cases found in the literature, both anatomists and clinicians have reached this conclusion by exclusion always however *cum grano salis.*

This somewhat critical discussion of the objective diagnostic expedients was necessary, in order that a stand might be taken on the frequently discussed question, as to the *relation of sudden deaths in infancy to hyperplasia of the thymus.* Such catastrophies happen much oftener during the first eighteen months of life than in adults. The autopsy in many of these cases gives a sufficient explanation of the death. The acute capillary bronchitis of sucklings plays a prominent rôle, and the surprising part of such deaths may be sufficiently explained by the lack in these cases, of subjective complaints and of objective observations.

Nevertheless, a number of such cases remain, which even the most careful autopsy does not fully clear up. When no long wasting illness has preceded the death, and a considerably enlarged thymus gland is found, we are apt to explain the sudden death as due to this cause, after the observations of Baginsky and Grawitz. Numerous published reports have given plenty of material for the thorough review of the question. Several years ago it was critically sifted. Since then, several new communications have been added to the literature, but without bringing out anything of value. These authors are of the opinion that hyperplasia of the thymus could cause sudden death by pressing upon the neighboring vital organs; either by sudden enlargement of the gland,

or by a particularly unfavorable position of the head, or by both. Some other far-fetched hypotheses have been advanced.

But here also, opinions differ, while one observer holds that the threatened organ is the trachea with the large bronchial tubes; another believes it to be the aorta that is in danger, or the pulmonary artery, or the superior vena cava and its two larger branches, or the veins of the lungs, or part of the heart, and last, the vagus, phrenic and sympathetic nerves. Most of these opinions are mere hypotheses, arrived at because of the topographical and anatomical relations of these various structures to the gland, and are scarcely supported by positive findings. As an objection to the above in a general way, one might answer, that hyperplasia of the thymus would act permanently and gradually rather than suddenly, if pressure was being exerted by it upon one of these important organs.

The occurrence of a sudden swelling has never been proven, and all other attempts at explanation cannot withstand criticism. Not even the theory that a critically narrowed upper thoracic aperture, might be still more narrowed by a sudden forceful extension of the head.

How can anyone imagine the thymus everting pressure of any importance at all, upon the neighboring organs? The absolute weight of the gland, which has I think, wrongfully played such an important part for so long a time, cannot be taken into consideration for the reason that the gland being bound to all the surrounding organs by connective tissue is in a manner hanging in place; and besides it could only act upon the organ upon which it lies when the body is in the supine position; and then with only a fraction of its weight.

A benignly enlarged thymus can only afflict the neighboring organs within the measure of its osmotic pressure and it will grow in the direction, obviously, of the least resistance. That it could compromise a normal trachea or normal large arteries is therefore excluded. We know that an aneurysm can erode bones. How is it possible then that so powerful a vessel as the aorta could be narrowed by pressure exerted upon it by the thymus, as is thought possible by some authors? (Lang, Cohn.)

In reality, pressure seams have been found on large thymi due to compression exerted upon them by the innominate artery and by the manubrium.

The large veins, possessing little tension, would appear to be in more danger; but is it at all probable that in the depths of the thorax an exchange pressure could take place, affecting the veins only, and not rather affecting the so easily compressed lungs! The veins mentioned by these authors lie below the "critical aperture" and besides, no sudden closure of the veins has been verified on post mortem.

A serious consideration must be given to those cases of narrowing of the trachea and bronchial tubes; which have been proven as such,

and which have been repeatedly observed since they were described by Somma; on account of their being analogous with the deformities of the trachea caused by struma, and with the cases of death caused by struma goitre.

Even if the healthy trachea of the newborn infant, according to the experiments of Tammassia and Scheele, can withstand much pressure, still, a trachea changed by disease, might show considerable softening of the cartilaginous ring, and in such cases as Marfan and Lange describe, might be compromised by a large thymus (Pfaundler). Still, such changes have never been proven and should they occur, we would expect a tracheo-stenosis long before death occurred.

A clinical picture of this kind does exist indeed, in the early suckling age and would best be called by the name chosen by Hochsinger, as:

Stridor Thymicus Infantum. This is a more or less severe tracheo-stenosis, which generally develops during the first weeks of life; but which sometimes is present at birth and which increases under excitement. This disease generally ends benignly, by the gradual disappearance of the symptoms, as the infant grows and the trachea becomes more firm. In very severe cases, courageous operators have undertaken a thymus plastic operation with success. This was first done by Rehn.

It would not do to mistake the foregoing picture for the so-called stridor congenitus. It differs from the latter in not being a pathological unity, but in being due to different causes (Breceli) one of which might be hyperplasia of the thymus.

Coming back to the former question, the attempts to explain unexpected deaths in childhood, from the local effects of hyperplasia of the thymus, must be looked upon as a failure, up to this time. Even the stridor thymicus, is an exceptionally chronic picture of illness and cannot be used as a support for the arguments of those who take that view.

A. Paltauf in 1889 was forced, on the strength of his own many experiences as pathologist, to the same conclusions. But he only declined to accept the false explanations; while he confirmed the fact of the coincidence of sudden death with the presence of an enlarged thymus, and gave it quite a new interpretation. According to him, the cause of sudden death, apparently impossible to explain, lies in a peculiar constitutional anomaly; which makes its possessor weak and less able to withstand attacks of illness, death being easily produced from trifling causes.

The corpses of those dying thus are characterized by the enlargement of lymph-nodes, tonsils and follicles at the base of the tongue, and of the intestines; swelling of the spleen and an enlargement of the thymus, the size of which varies; particularly at an age when it has generally disappeared. To all this must be added a narrowness

and delicateness of the aorta and the rest of the arterial system, signs of acute dilatation of the heart with a soft pale heart muscle; which occasionally shows signs of degeneration. These are, according to Paltauf, the signs of the lymphatic-chlorotic constitution; which one calls in short, the *status lymphaticus*.

It should be remembered that the hyperplasia of the thymus is only a symptom of the above-mentioned condition, and that it does not act locally as a cause of death; but only marks its bearer as a particularly vulnerable individual. This doctrine has at last been accepted by the clinicians. Among other pediatrists, Pott and Escherich have chiefly helped it gain popularity.

Kassowitz is fully justified in calling attention to the spasm of the larynx and particularly to the expiratory apnœa; which plays a rôle in most of the sudden deaths occurring in early childhood; for this is indeed the time when neurotic conditions and rachitis, together with tender age, render the little patient liable to attacks of laryngeal spasm; and this is the time when these shocking deaths occur.

The acceptance of the theory, even when the conclusion is missing; when the death has occurred at the time of the first seizure is also worthy of notice; and Thiemich essentially puts himself on record as holding similar views.

But why do only certain children succumb to such attacks, and how shall we explain the sudden deaths which occur sometimes in older children, and even occasionally in adults ill with infectious diseases of a fulminating malignancy? How shall we explain the question lately raised by Feer, as to death from eczema? As clinicians we cannot agree with Richter's sceptical views and must therefore consider the hypothesis of Paltauf as a considerable advance, in spite of the above arguments not being quite convincing.

The *status lymphaticus* which after the excellent teachings of Escherich, presents a familiar picture to every pediatrist is a useful link, binding together all these clinical experiences and theories. This condition is generally found in well nourished, small, pale, somewhat puffed up looking children. Their superficial glands, spleens and the glands of their necks are enlarged. Dulness on percussion over the thymus is often easily made out. Rachitis and scrofulosis are often present. A large contingent suffer from prurigo, chronic eczemas and spasm of the larynx. In some of these cases trifling therapeutic encroachments, or acute infections where ordinarily the prognosis would be good, end in death. Escherich has tried to explain this constitutional anomaly as analagous to the strumous cachexia present in so many cases of exophthalmic goitre. He claims that this condition is a consequence of the disturbance of the functions of the thymus. Many experiments and inquiries, particularly the observations of Svehlas

seem to support these explanations; but of course, they all remain debatable. One might point out the finding of an enlarged thymus in many chronic constitutional diseases; particularly in cases of endogenous poisonings; as an expression of defense on the part of the organism; but after all these findings open up the possibility of accepting the status thymicus as a similar poisoning from which the organism protects itself by hyperplasia of its reserve forces; the lymphatic apparatus.

II. The *absence of the thymus* does not seem to play much of a rôle in the pathology of children. It is hardly worth mentioning. If absent, it is in those cases where there is a deeply inherent deformity of the rest of the body; which at once precludes a long life. The few cases on record of absence of the thymus belong to the older literature and have no significance.

III. *Atrophy of the thymus* is the normal finding in cadavers after severe chronic illness, and also in the cadavers of badly nourished individuals. The attempt therefore of Seydels to make the atrophy of the glands, forensically, a sign of exhaustion; as for instance, death from inanition, has to be declined, on account of its being open to a great variety of explanations.

IV. *Thymitis.*—Acute inflammation of the gland with the formation of abscesses was formerly seemingly frequently established by post mortem, the examiners having mistaken another fluid for pus. However, genuine abscess of the thymus does happen now and then in the course of pyæmic processes in young children. One thing I have noticed is that after a severe tracheotomy, followed by a foul condition of the wound, the thymus never appears affected. Abscess never forms.

V. *Tuberculosis of the thymus* is also of no clinical importance. On the dissecting table, one sometimes finds it in cases which have succumbed to an acute miliary tuberculosis or a chronic tuberculosis. If careless, one might easily be deceived and think the case primarily one of tuberculosis of the thymus, if caseated lymph-nodes are fused together with it.

VI. *Syphilis of the thymus* is only of theoretical interest. According to Schlesinger this gland is affected in cases where the disease is general, in about 40 per cent. of the cases. The most frequently found manifestation is a diffused cellular infiltration; which is followed by shriveling. Occasionally one finds gummata which sometimes soften considerably, but the characteristic abscess first described by Dubois, has nothing to do with the syphilitic lesion and is only to be considered, according to Chiari, as systemic. The multifold appearances of this condition were well known to Bednar. Hæmorrhages also, are described by a few authors as characteristic of syphilis.

VII. *Tumors of the Thymus.*—Since the careful separation of the thymus from the mediastinal lymph-glands, it is very rare to find a case of malignant disease of the thymus alone. The differential diagnosis for proof of primary cancerous disease of the thymus is practically impossible; for in all these cases the symptom-complex is that of a mediastinal tumor of the lymphosarcoma variety. Finding the Hassalls corpuscles and polymorphic cells in a tumor of the thymus, directly behind the sternum, has been thought proof by some, that the tumor developed primarily in that gland, but even this is not sufficient proof.

A few cases of lymphæmia with primary hyperplasia of the thymus have been reported.

DISEASES OF THE CIRCULATORY SYSTEM

BY

Dr. C. HOCHSINGER, of Vienna

TRANSLATED BY

Dr. MAURICE OSTHEIMER, Philadelphia, Pa

I. ANATOMY AND PHYSIOLOGY OF THE CIRCULATORY APPARATUS

1. DEVELOPMENT OF THE HEART AND LARGE VESSELS

A. The Heart.—The primary cardiac tube, which lies alone in the cervical region, behind the last visceral arch, possesses two ends, a posterior for the reception of venous blood, and an anterior end which serves to supply the blood to the body. In amniotic vertebrates, the heart, which consists of two ventricles and two auricles, arises gradually by constrictions and twists of this tube in many places, as well as by the formation of internal septa. The cardiac tube, originally straight, becomes an S-shaped loop, differentiated into three parts: the upper or aortic portion with the bulbus, the lower venous portion, and the middle or ventricular portion. This central part soon bends upon itself, into the shape of the stomach, so that what was originally the posterior, venous portion of the heart is directed more dorsally and the arterial portion more ventrally. By the lateral outgrowth of blind sacs from the venous portion, to both right and left, the beginnings of the auricles develop, while that part of the central portion corresponding to the greater curvature is divided in its centre by a shallow groove, the sulcus interventricularis.

The earliest processes of ventricular division appear between the fourth and fifth fœtal weeks, when a perpendicular septum grows up inside, from the groove just mentioned, while a transverse constriction, canalis auricularis or auricular canal, is formed between the atrium and the ventricular portion. The atrioventricular valves develop within the auricular canal. By the eighth week this upward growing interventricular septum has reached the auricular canal, so that one common atrium and two ventricles are formed at this time. The single atrium freely communicates with each ventricle through the right and left atrioventricular ostia. Only then does the truncus arteriosus communis divide into aorta and pulmonary artery, by the development of two winglike septa which gradually grow together to form the septum

trunci, dividing the originally single tube into two tubes, lying parallel to one another like the barrels of a double-barreled gun. Now, as the septum trunci is growing downward in the same direction in which the interventricular septum grows upward, it penetrates the ventricular cavity until both septa meet, with the final result that the aorta communicates directly with the left ventricle and the pulmonary artery with the right ventricle. The separation of the single truncus arteriosus, however, first occurs in its lowest part, while further upward pulmonary artery and aorta still remain united, forming later the ductus Botalli.

The development of the auricular septum forms the last stage in the partition of the cardiac cavities. A septum grows from above and behind, ending within the cavity in a concave border. To the right of this fold opens the superior vena cava, while the inferior vena cava enters the auricle just beyond the edge of this fold. In its centre remains an opening, the foramen ovale. Inside arise the valve of the foramen ovale and the Eustachian valve which direct the fœtal blood stream into the auricles (see Fœtal Circulation, page 437).

The valves on the venous ostia (atrioventricular valves) grow from tumor-like thickenings of the ostial edge and connect the ventricles by bundles of muscle which are grouped with the subsequent papillary muscles (Bernays). According to Röse, this is true only of the median valves, while the lateral valves arise directly from the ventricle wall by the separation of true muscle which is transformed into connective tissue later.

The valves upon the arterial ostia develop from peculiar cushions of endothelium, are at first coarse and thick and later become delicate, acquiring thin walls.

B. The Arteries.—The aortic arches, originally but a single one, become multiplied into five for each side with the development of the five visceral arches, and join to form a common trunk directed downward (Rathke). In human beings the two upper visceral arches on each side atrophy first. With the division of the truncus arteriosus communis into aorta and pulmonary artery the lowest arch on the right side becomes the pulmonary artery, and therefore arises from the right side of the heart, while the arch on the left becomes transformed partly into the ductus Botalli and partly into the main branches of the pulmonary artery. The middle arch on the left side becomes the permanent aortic arch, into which the ductus Botalli empties; the middle arch on the right becomes the right subclavian artery. The uppermost arch on each side becomes the origin of the carotid artery.

C. The Veins.—It should be noted, in regard to the venous system, that four cardinal veins are formed, an anterior and a posterior for each side, and that, before emptying into the heart, the two on each side unite to form a common trunk, the duct of Cuvier. The

large veins of the body, the superior and inferior venæ, arise by the formation of numerous anastomoses and divisions of these two sets of cardinal veins and empty into the right ventricle, as will be shown later.

2. THE FŒTAL CIRCULATION (Fig. 87)

The circulation of the blood in the fœtus differs from normal extra-uterine circulation in that the systemic and the pulmonary circulations are not separated in the fœtus and that, excepting the umbilical vein which contains pure arterial blood, most of the fœtal vessels carry mixed blood.

The fœtal circulatory communications, all of which atrophy after birth, are the following:

1. *Ductus venosus Arantii.*—This is a continuation of the umbilical vein, *i.e.*, a communication from the latter to the liver. The umbilical vein, rising from the placenta, divides below the liver into two branches, the longer of which reaches the superior vena cava as ductus Arantii, the shorter one going to the left branch of the portal vein. Umbilical vein and ductus venosus Arantii atrophy to form the ligamentum teres.

2. *Umbilical Arteries.*—They arise from the hypogastric arteries of the fœtus, pass out of the child's body through the umbilicus, surround the umbilical vein in their twisted course and break up into branches in the placenta. Before birth they carry blood from the fœtus to the placenta; after birth they atrophy to form the ligamenta vesicalia lateralia.

3. *Ductus arteriosus Botalli.*—In the fœtus this furnishes the direct blood vessel connection between pulmonary artery and aorta. The blood is carried through it from the right ventricle directly into the systemic circulation. The ductus arteriosus Botalli arises just where the pulmonary artery divides, and runs obliquely upward to the concavity of the aortic arch. This vessel atrophies soon after birth. The atrophy is the result of expansion of the lungs with inspiration, which causes the blood to be sucked from the pulmonary artery into the lungs, thoroughly filling the branches of the pulmonary artery. Under normal conditions a sound can no longer be passed through the ductus arteriosus Botalli even a few days after birth; in extra-uterine life, from thrombosis and the growth of tissue arising from the blood vessel walls, it forms a solid cord, the ligamentum arteriosum.

4. *Foramen Ovale.*—This is an egg-shaped opening in the interauricular septum. The blood from the inferior vena cava, which flows into the right auricle, chiefly arterial blood, is directed by a projecting semilunar membrane, the Eustachian valve, toward the foramen ovale, through which most of it reaches the left auricle. The final closure of this foramen occurs toward the end of the first year of life, by the semilunar valve gradually growing forward over the upper edge of the oval opening.

The blood stream in the fœtus takes the following path: first, arterial blood from the placenta is carried by the umbilical vein to the liver of the fœtus. Before reaching the liver, the blood stream divides

FIG. 87.

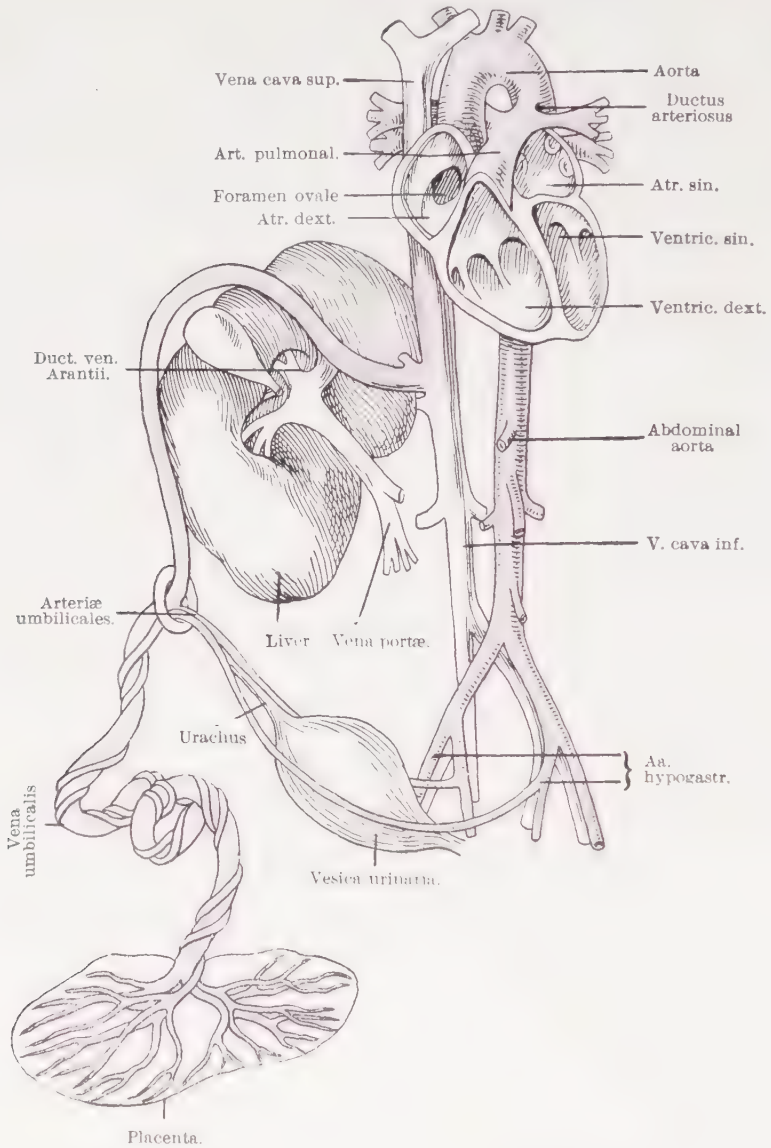


Diagram of the fœtal circulation, from Heitzmann's "Anatomischer Atlas."

into two branches, one going through the ductus Arantii into the inferior vena cava, the other joining the portal vein directly, also finally reaching the inferior vena cava through the hepatic veins, mixed with the blood of the portal vein. Before the inferior vena cava enters the right auricle, it carries mixed blood from the following sources: arterial

blood from the umbilical vein, venous blood from the portal vein, and venous blood from the lower half of the body.

The great part of the blood from the inferior vena cava is carried from the right auricle, as a result of the peculiar position of the Eustachian valve already described, into the left auricle; thence it enters the systemic circulation, and through the hypogastric and umbilical arteries of the fœtus, it again reaches the placenta.

The blood from the superior vena cava is sent from the right side of the heart through the pulmonary artery into the lungs on the one side, and on the other, through the ductus Botalli into the aorta.

There is, nevertheless, a decided difference in the distribution of the blood within both halves of the heart, although both receive mixed blood. The great mass of the blood in the left half of the heart arises from the inferior vena cava, only a small part coming from the lungs. The right side of the heart contains blood poor in oxygen, as it receives an overwhelming quantity of almost pure venous blood from the superior vena cava. The blood carried back into the left side of the heart by the pulmonary veins is also pure venous blood, since it receives no oxygen in the lungs of the fœtus.

Besides, the fact that the upper half of the fœtal body receives blood richer in oxygen than the lower half is settled. The descending aorta contains venous blood carried into it by the ductus Botalli, so that the arteries which leave it after it has united with that vessel carry blood which is not so pure as that found in the arteries supplying the upper half of the body, which arise from the aorta above the ductus Botalli.

Complete separation between the systemic and the pulmonary circulations is first brought about by birth. But preparations have been made for this toward the end of pregnancy; the foramen ovale and the ductus Botalli become smaller in lumen during the last months of pregnancy, and second, the growing lungs demand more blood toward the termination of pregnancy than in the earlier months. Therefore, toward the end of pregnancy, a decreased amount of blood is carried into the left auricle by the inferior vena cava, and a smaller quantity of blood is sent through the ductus Botalli into the descending aorta.

At the moment of birth the placental circulation is interrupted and in its place pulmonary respiration occurs in the child independently. The umbilical vein no longer carries blood into the right auricle; besides, the right ventricle must supply more blood to the unfolding lungs. As a result, a decided diminution in blood pressure occurs in the right side of the heart, which is also a cause for the closure of the ductus Botalli. Now, however, the lungs have withdrawn almost all the blood from the right side of the heart and have returned it, arterialized, into the left auricle. Here, in opposition to the right auricle, in which blood

pressure has become less, the blood pressure must increase. These changes in the circulation of the blood, occurring independently, prepare for the obliteration of the foramen ovale; the valves of the foramen ovale lie with their edges close to the upper free surface of the foramen and gradually fuse with it. Complete separation of the systemic and pulmonary circulations is made perfect after birth by the interruption of the blood current through the ductus Botalli and ductus Arantii on the one hand, and through the foramen ovale on the other.

3. PHYSIOLOGY OF THE CHILD'S HEART

The child's heart, in comparison with the rest of the body, is relatively larger in size than that of the adult, and the lumina of the large arteries are relatively greater. The younger the child, the more marked are these relations. A result of this is the fact that the blood pressure in the child's aortic system must be lower than in that of the adult. These conditions change with increasing relative narrowness of the arterial system toward puberty. While, in early infancy, the transverse diameter of the large arteries (carotid and subclavian) is very great in comparison with the length of the body and the size of the heart, this decreases relatively very markedly from the seventh to the fifteenth year, when a decided growth in the size of the heart occurs; for at this time subclavian and carotid arteries are relatively smaller in size than they are at any other time during life (Beneke).

Expressed in figures, according to Baginsky, the volume of the heart is to the lumen of the ascending aorta of the child as 25 is to 20; just before puberty, as 140 is to 50; right after puberty, as 290 is to 61.

According to Beneke, the volume of the heart increases most markedly in the first year of life; from then to puberty it diminishes, in comparison with the size of the body, but still without losing its preponderance when compared with the other organs of the body. According to von Dusch, the size of the heart is relatively greatest in the newborn infant and drops during the first and second years of life rapidly to a minimum in comparison with the whole mass of the body, to increase again from the third to the seventh year.

In newborn infants the thickness of the muscle walls of the left and right ventricle differs only slightly (0.44 to 0.48 left, to 0.34 to 0.44 right, Bednar), while the weight of the left is to that of the right as 1.3 is to 1 (Engel), compared with 2.62 to 1 in the adult. At six years the thickness of the wall of the right ventricle is not much greater than in the newborn (3 to 4 cm.), while that of the left ventricle reaches 4 to 8 cm.

Pericardial fat is absolutely absent in the newborn infant, is but scantily present during childhood and only develops in large amount after puberty (W. Müller).

The ostia of the right side of the heart in childhood are wider than those of the left (see Bizot's table below).

THE LENGTH, BREADTH, AND THICKNESS OF THE HEART IN CM.

Age.	Length.		Breadth.		Thickness.	
	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.
1 to 4 years	5.14	5.10	6.09	5.83	2.44	2.28
5 to 9 years	7.94	6.00	7.44	6.54	2.89	2.55
10 to 15 years	7.67	6.59	8.35	7.04	3.16	2.84

THE THICKNESS OF THE LEFT VENTRICLE IN CM.

Age.	Base.		Middle.		Apex.	
	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.
1 to 4 years	0.67	0.57	0.65	0.36	0.43	0.46
5 to 9 years	0.74	0.69	0.86	0.70	0.58	0.52
10 to 15 years	0.81	0.74	0.86	0.72	0.72	0.54

THE WIDTH OF THE VENOUS AND ARTERIAL OSTIA IN CM.

Age.	Left ventricle.		Right ventricle.		Aortic ostium.		Pulmonary ostium.	
	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.
1 to 4 years	5.68	5.86	6.86	6.09	3.83	3.62	4.20	3.83
5 to 9 years	6.77	6.30	7.67	7.42	4.13	3.88	4.42	4.17
10 to 15 years	7.14	7.16	8.80	7.67	4.81	4.28	5.05	4.60

The increase in growth of the size of the heart is not proportional to the growth of the body, but bears a steadily decreasing ratio to it, according to W. Müller, whose statements are founded upon 1481 autopsies.

The volume of the heart of the newborn infant, taken absolutely, is about 23 c.c. and increases to about 100 c.c. by the seventh year. Between the seventh and fifteenth years the heart no longer increases so considerably in volume, as it reaches on the average only 140 c.c. at the end of the fifteenth year. Thus the size of the heart is relatively smallest just before puberty, and during the development of puberty it again rapidly increases in bulk.

The weight of the heart in the newborn infant is, according to K. von Vierordt, 0.89 per cent. of the body weight (absolutely, on the average, 24 Gm.; according to Bednar, 18–20 Gm.) as compared with 0.52 per cent. in the adult; *i. e.*, considerably higher, in relation to the total body weight, than in the adult. This favorable relation diminishes gradually as the years of life increase. While the adult's total body weight at the end of his development has reached nineteen times the weight of the newborn infant, the heart has grown to be only fifteen times as heavy as it was, so that the relation of the weight of the heart to that of the body is never again as favorable as it was in earliest infancy.

It is a remarkable fact that the increase in the circumference of the heart, during its growth in the first years of life, is not proportional to its development in size and increase in weight. The circumference of the heart does not change much more in the first five years of life, in spite of the heart's increase in weight. This fact has been corroborated by the circumference estimations made by Bednar and Rilliet and Barthez.

Rilliet and Barthez undertook to measure the circumference of the heart at the base of the ventricles, with the heart filled and empty, in the cadavers of 193 children which came to autopsy. These investigations are especially valuable because many of the children died so young. Thus 51 cases were under $2\frac{1}{2}$ years; 29 between 3 and $3\frac{1}{2}$ years; 21 from 4 to $4\frac{1}{2}$ years and 14 from 5 to $5\frac{1}{2}$ years; so that 115, out of 193 children, had not yet reached the age of 6 years.

Rilliet and Barthez came to the following conclusions:

1. The circumference of the heart does not increase relatively with age; it is almost the same from 15 months to $5\frac{1}{2}$ years; from then on it increases regularly until puberty.

2. The distance from the base to the apex of the heart, anteriorly, is almost exactly one half of the entire circumference at the base of the ventricles.

3. The greatest thickness of the wall of the right ventricle varies little with regard to age; up to the sixth year it measures on the average 2 mm., in later years usually from 2 to 4 mm.

4. The greatest thickness of the wall of the left ventricle up to the sixth year is not quite one, later commonly more than one cm.

5. The size of the right venous ostium remains almost the same up to the fifth year; from this time to the tenth year it increases slightly, but only grows somewhat in the tenth year.

6. The left venous ostium, always smaller than the right, increases a trifle more regularly than the right from year to year.

7. The aortic ostium hardly grows at all from 15 months to 13 years.

8. The pulmonary ostium, on the contrary, grows considerably from the sixth to the eighth year, so that, although it was just as large or hardly larger than the aortic ostium before that time, it is much larger than that opening afterward.

Looking back over these conclusions shows that the heart of the young child has great advantages over that of all other ages, especially of later childhood. That the circumference of the heart does not increase through the first five years, although the size and weight of the heart do, shows that the heart muscle steadily becomes bulkier and stronger during this time. It follows from this that the increase in the circumference of the heart during this time is due, not to increase in the cavity, but to continual increase in the muscle mass. After the end of the first

five years, the increase in the size of the heart is accompanied by considerable dilatation of its cavities at the same time.

The long time that the size of the ostia remains stationary also speaks in favor of the child's heart and its working ability, which is relatively small in spite of its bulky musculature. This circumstance proves that the obstacles which the cardiac muscle has to overcome, upon the entrance and exit of the blood stream through the ostia, are incomparably slighter in earliest infancy than at more advanced periods of life.

From all of this it results that functional disturbances of the heart muscle occur much less frequently in childhood than in adults, and that injuries of general significance will exert an influence upon the child's heart very much later than upon the heart of an adult man. The child's heart, also, as opposed to pathologic changes in its valvular apparatus, has more material to make up for disturbances in compensation dependent upon the performance of its work; *i.e.*, it always has something in reserve.

Blood Pressure.—The blood pressure is best measured by Gärtner's tonometer, provided with smaller finger compressors and rings, suitable for children. An exact estimation in infants is not always possible because of the small size of the finger phalanges, the thick cushion of fat upon them and the difficulty in adapting the rubber compressors. Trumpp found the average estimate in a healthy infant to be 80 mm.

The following figures are to be considered normal, according to Kolossowa.

1– 2 years	80– 85 mm.
3– 4 years	85 mm.
5– 7 years	90– 95 mm.
8–10 years	95–100 mm.
11–13 years	100–110 mm.

Considerable diminution in blood pressure gives an unfavorable prognosis, especially in diphtheria.

The relative mass of blood in the newborn infant is the same as in the adult (Robin and Hiffelsheim). The work of the heart in the unit of time, taken absolutely, is, according to Vierordt, 20 times as great in the adult as in the newborn infant; relatively, it is greater in the child than in the adult. The mass of blood which, in the unit of time, passes through the unit of weight of the organism is 379 c.c. in the newborn infant; 306 c.c. in the child of 3 years; 246 c.c. in one 14 years old, and 206 c.c. in the adult.

CONDITIONS OF THE BLOOD VESSELS

Beneke has recognized as the cardiovascular type of childhood a small cardiac cavity with wide body arteries, a condition which becomes reversed after puberty.

The volume of the heart grows to 12 times its size from birth to puberty; the circumference of the aorta grows to only 3 times its original size. During earliest childhood the relation of the volume of the heart to the circumference of the aorta is as 25 is to 20; at the time puberty develops, it is as 140 is to 50; at full maturity it is as 260 is to 61. The ostia grow only slowly and remain of about the same size during the whole of childhood.

The lumen of the large arteries of the upper half of the body, the carotids and subclavians, in early childhood is greater than that of those of the lower half, iliac arteries, a condition which is dependent directly upon the energetic brain development.

In the adult the relation of the lumen of the veins to that of the the arteries is as two is to one; according to Alix, in early childhood, both vessels measure the same.

The walls of the child's veins are more resistant than in the adult.

In looking over the above anatomic peculiarities of the circulatory apparatus in childhood, three essential clinical characteristics will be noted: (1) diminished blood pressure, (2) rapid circulation and (3) rapid pulse, without other signs.

II. GENERAL SYMPTOMATOLOGY AND DIAGNOSIS

The symptomatology of heart diseases in childhood is somewhat simpler than in later life. Secondary conditions in remote portions of the body, such as congestion of the parenchymatous organs or dropsy, are almost entirely absent in the heart affections of early childhood, because of the extraordinary tolerance of the child's heart. Recovery from acquired endocarditis is also a much more frequent result in child hood. Auscultatory signs play the chief part in the symptomatology. Congenital as well as acquired heart lesions may exist in children a long time without any change in the percussion dulness.

The most important *auscultatory changes* in the child's heart are the *murmurs*, which are more characteristic than in adults. While the same value in diagnosis is attached to exocardial murmurs in children as in adults, endocardial murmurs show a varying relation, in that infancy is almost entirely free from the so-called accidental heart murmurs. In the second and third years of life, too, so-called anæmic murmurs are very rare. Hochsinger, Soltmann, Délabost and Romberg have accepted the complete absence of accidental (so-called anæmic) heart murmurs in the first years of life, yet this is contradicted by Thiemich, von Starck, Abelmann, Rheiner, Methling, Jacobi, Heubner, Swarsenski and Looft, who concede only their great rarity in early childhood.

The systolic murmurs by far outnumber diastolic and presystolic murmurs in frequency; besides systolic murmurs are very often only added to the first heart sound, while diastolic murmurs almost always

replace the second heart sound wholly. The temporary concealment of heart murmurs by accelerated respiration and râles is much more frequent in children than in adults. On the contrary, temporary disappearance and return of murmurs (disappearance when at rest, recurrence with exertion) are only noticed in children exceptionally. Cardiac murmurs as the result of acquired heart affections are heard with more difficulty in early childhood than in older children and adults; while in congenital heart lesions very loud murmurs have already been observed, even in infancy. Only the latter are well transmitted to the back. Thrills, which are palpable heart murmurs, are more apt to accompany the heart action of a child than that of an adult, because the child's thin chest wall oscillates more easily. Heart murmurs dependent upon acquired affections appear chiefly at the mitral valve; those caused by congenital lesions more especially at the pulmonary ostium. In the former the point of maximum intensity of the murmurs will be at the apex; in the latter, in the second intercostal space to the left of the sternum. Not always, as is the case occasionally in congenital cardiac anomalies, is the point of maximum intensity the same as the point of origin of the murmur, for several murmur-producing causes may be located at different places within the heart, and by transmission to one spot, produce an especially loud acoustic impression there. The point of origin of a heart murmur can sometimes be determined by observing the transmission of the murmur to the back. If, in little children, murmurs are transmitted to the lower left side of the back better than to the upper, then most probably the murmur is due to a change at the venous ostia. When the opposite is true, the origin of the murmur with approximate certainty is at the base of the heart, *i.e.*, at one of the arterial ostia.

Organic Endocardial Murmurs.—These occur in acquired congenital heart lesions of children. The acquired heart diseases are inflammatory diseases of the endocardium and acute dilatation of the heart, which lead to relative insufficiency of the venous valvular apparatus. The timbre of the murmur is as a rule higher and shriller in children than in adults and the murmur is transmitted further over the anterior chest wall.

The murmurs of acute dilatation of the heart in children, occurring sometimes in the course of scarlatinal nephritis, are accompanied by dyspnœa, pain in the chest and a tendency to collapse. The cardiac dulness becomes very much increased laterally, the pulse very weak and frequent. In acute endocarditis, on the contrary, at the time of the first appearance of murmurs, signs of dilatation of the heart are usually absent, as are the other severe accessory symptoms just mentioned.

Presystolic Murmurs, so important in the diagnosis of mitral stenosis in later life, are almost entirely absent during the first years

of life, as are accentuation of the second heart sound at the aortic area and excessive tension of the radial pulse. On the other hand, accentuation of the second sound at the pulmonary area is observed in the first months of life, especially with congenital heart lesions, and is of exceptional value in diagnosis on account of the slight accentuation due to closure of the semilunar valves under normal conditions, a fact first established by Hochsinger. Just as valuable is the diminution or absolute inaudibility of the second sound at the pulmonary area, an infallible sign of pulmonary stenosis.

Organic endocardial murmurs may be simulated in childhood by cardiopulmonary murmurs, intrathoracic venous murmurs and rapid respiration. Statements of the occurrence of accidental cardiac murmurs in early childhood are for the most part founded upon mistakes made in hearing cardiopulmonary murmurs (systolic vesicular breathing, according to Wintrich). This systolic murmur, first recognized by Hochsinger in childhood, arises in the portions of the lungs nearest to the heart and is due to the entrance of air into the edges of the lungs during inspiration, this air being changed regularly with systole and diastole. Rapid respiration and accelerated heart action, conditions which are present in childhood especially, are necessary for the appearance of this phenomenon. These murmurs are always systolic, very harsh, sometimes completely concealing the first sound of the heart, at other times simply appended to it. They are differentiated from organic murmurs only by their variability above mentioned, which, however, is not always easily discoverable. They become louder when respiration pauses during inspiration; weaker or absolutely wanting when one's breath is held during expiration. They occur especially frequently after the third year, yet W. Freund, Rheiner and Hochsinger have noted them in infants. In older, easily excitable children, cardiopulmonary murmurs are especially frequent; Potain's so-called "souffles des consultations" in excited patients are nothing other than cardiopulmonary murmurs. Slight pressure made by the stethoscope at the point of origin of the murmur increases it; with more pressure the murmur is lost. Cardiopulmonary murmurs occur most frequently over the left ventricle, much more rarely at the apex, and very rarely over the aorta and auricles (Délabost).

Délabost explains the so-called accidental murmurs of children, taken altogether, as cardiopulmonary murmurs, and refers the absence of the accidental murmurs in early life to the scanty covering of the heart by lungs during the first months of life, a fact settled by Délabost, as well as by West, Durand, Soltmann, and Hochsinger earlier. The few cases of so-called anæmic or accidental heart murmurs in early childhood, noted in literature, appear in a different light from the standpoint of the origin of cardiopulmonary murmurs. Systolic mur-

murs at this period of life, if they are not dependent upon organic intracardial affections, are usually considered extracardial, arising in the edges of the lungs when filled during inspiration, and not as true accidental heart murmurs. Only those murmurs which appear as the result of aperiodic vibrations of the valves following nutritional disturbances of the cardiac musculature, without any anatomic change, are regarded as true accidental heart murmurs.

With low blood pressure and very rapid respiration, organic heart murmurs may also completely disappear, to reappear again when respiration and pulse-rate diminish, as they do after the administration of digitalis. Nor must the fact be overlooked that myocarditis in children can produce systolic murmurs similar to those of endocarditis, only these murmurs are less constant than the murmurs of endocarditis and may appear with symptoms very like those due to cardiopulmonary murmurs; but the first sound is hardly ever completely concealed by them.

According to Kimla and Scherer there must be great hæmorrhage to have produced murmurs in newborn infants.

Steffen, by pressure with the stethoscope on the anterior chest wall, could produce a weakening of the heart sounds in rachitic children with yielding chests, besides, he could change the heart sounds into murmurs; while Henoch, by pressure at the pulmonary area artificially caused murmurs there. Such murmurs are not to be considered accidental heart murmurs, but artificially produced compression murmurs. In high-grade rickets, in which the junction of ribs and costal cartilage cannot be broken inward, the pulmonary artery may be compressed by bending the ribs in, and a constant systolic murmur can be heard, even without the pressure of a stethoscope, which is also not an accidental but a compression murmur.

In the course of severe pulmonary affections and the infectious diseases, even in the earliest periods of life, systolic murmurs may arise in the pre-agonic stage, which are due, not to changes produced by endocarditis, but to relative dilatation insufficiency of the atrioventricular valves, or to paralysis of the heart, but these murmurs may be mistaken for accidental murmurs.

Arterial Murmurs.—Small children during the first 2 to 3 years have no peculiar sounds in the arteries of the neck. Such sounds are found in older children, but may be changed into murmurs by moderate pressure with the stethoscope. The occurrence of murmurs in the arteries of the neck is only of value in diagnosis in childhood if the murmurs are transmitted from the heart and are recognizable as such, a condition which is observed very frequently in congenital heart lesions.

Venous Murmurs.—Venous murmurs are very frequent in all periods of childhood. They can be heard over the chest, on both sides of the sternum, when they occur in the innominate veins. Even more

frequent are murmurs in the veins of the neck, with or without murmurs in the innominate veins. Older children with anæmic heart murmurs always show murmurs in the veins of the neck, while children with true heart diseases often have such murmurs also. In the combination of cardiac and venous murmurs, it should be the rule that endocardial murmurs localized at the pulmonary area are to be considered accidental, while a loud cardiac murmur at the apex, without any murmur in the pulmonary area, shows endocarditis almost without exception, in spite of the simultaneous presence of a venous hum. Murmurs in the veins of the neck do not occur in healthy children if one is sufficiently careful, during auscultation, not to extend the neck too far or to press too hard with the stethoscope.

Venous murmurs which are especially limited to the right half of the chest occur in the right innominate vein, which is very commonly the seat of a murmur in anæmic, particularly tuberculous children (infants also); the left innominate vein shows no auscultatory anomalies. Possibly this venous murmur arises in the superior vena cava and is transmitted into the right innominate vein, the direct prolongation of the vena cava, while the left innominate vein, branching off at an angle from the superior vena cava, remains untouched by the murmur. The cases of unexplained systolic heart murmurs, noted by Gregor and Marfan, are to be regarded as innominate murmurs. They very frequently give the impression of a systolic murmur of long duration, since they are decidedly increased when the aorta fills with systole; whether rhythmical compression of the right innominate and vena cava, due to the filling of the aorta, occurs here or not, may be left undecided.

The venous murmur heard by Eustace Smith above the manubrium sterni depends, according to Smith, upon compression of the veins by bronchial glands. When the child's head is extended far backward a venous murmur is heard with the stethoscope placed over the manubrium sterni; if the child's head is moved forward the murmur becomes weaker; when it reaches its normal position the murmur disappears. The occurrence of this venous murmur with hyperplasia of the bronchial glands is dependent upon forward movement of the trachea as the result of overextension of the neck, so that the glands lying at the bifurcation of the trachea are shoved forward and pressed against the innominate veins. Hochsinger notes that this murmur is very frequent, in infancy especially, and also in children in whom there is no suspicion of enlargement of the bronchial glands. Hochsinger has found this murmur strikingly frequent in children with hyperplasia of the thymus gland, and with dulness noted over the manubrium sterni, dependent upon the presence of this gland. It always arises from pressure upon the innominate veins, whether hyperplasia of the bronchial glands or thymus gland exits or not.

THE NATURE AND FREQUENCY OF HEART AFFECTIONS IN CHILDHOOD

The numerous changes in the musculature and valvular apparatus of the heart which are caused by atheroma of the arteries, the use of alcohol and tobacco, mental and physical overwork, are almost entirely absent in the first years of life. It results necessarily, therefore, that acquired affections of the arterial ostia in the form of valvular stenosis and insufficiency, or primary myocardial affections must be rare in childhood. Almost without exception the cause of such changes in childhood is pericarditis or endocarditis as a result of the infectious diseases, which soon produce acute, subacute or chronic changes in the valves, ostia and myocardium; but these are not among the very frequent diseases of childhood. If we add to this the well-known fact that acquired heart affections are but rarely found at the ostia of the right side of the heart, and then only as remains of fœtal inflammatory processes, complicated by recrudescent endocarditis, it is easily understood that the occurrence of acquired heart disease is decidedly restricted in early childhood, being limited entirely to inflammatory changes of the left venous ostium.

Samson, from his observations, considers the frequency of heart diseases in childhood as 3 is to 500 ($\frac{1}{2}$ per cent.). Among 227 children treated for severe internal affections resulting from the infectious diseases, 38 had cardiac disease (16.7 per cent.).

Samson divided 131 cases of heart affections in children (100 of them his own observations) into groups according to age, as follows:

Under 1 year	4 children
in 2nd and 3rd year	5 children
in 4th year	7 children
in 5th year	8 children
in 6th year	15 children
in 7th year	14 children
in 8th year	14 children
in 9th year	17 children
in 10th year	18 children
in 11th year	23 children
in 12th year	9 children
Total	131 cases

In this table 24 cases occurred in the first five years of life, 18.3 per cent. This includes both children with congenital and acquired heart lesion. Cassel found, among 20,000 sick children, 107, about $\frac{1}{2}$ per cent., with heart affections (the sexes being equally divided), of which 26 were congenital.

The most important clinical signs for *differentiating between congenital and acquired heart affections* in children are the following:

1. Loud, harsh and musical heart murmurs, with normal or immaterially increased dulness, occur in small children with congenital lesions only. Acquired heart affections, arising from inflammation, with very loud heart murmurs, without exception show in small children large areas of dulness also. With combined congenital malformations, the cardiac hypertrophy may be increased by the mutual relations between the separate anomalies.

2. Heart murmurs with large areas of cardiac dulness and weak apex-beat point to congenital changes in small children. Dulness is increased on the right side of the heart, while the left side is but slightly changed. Acquired endocarditis of children is accompanied by accentuation of the apex-beat since the left side of the heart is most affected; only later is dilatation of the right side added, without changing the increased strength of the apex-beat.

FIG. 88.

Diagrammatic drawings, about one-fourth natural size, of the radiographic relations of the normal central shadow of the chest in infants.—The relations of the size are as true to nature as possible, but the outlines and shadings are semidiagrammatic. The single vertebræ are not drawn separately, but as the united shadow of the vertebral column. The shadows of the ribs and lungs are also omitted. These are addorsal photographs, in which the apex of the heart appears to the left. In *a* the cervical position of the central shadow of the chest is completely covered by the shadow of the vertebral column; in *b* this shadow stretches beyond that of the vertebral column, because of a somewhat larger thymus gland.

3. The absolute absence of murmurs at the apex, when they are clearly audible over the ventricles and at the pulmonary area, is always of great value in differential diagnosis, pointing to a defective septum or pulmonary stenosis rather than to acquired endocarditis.

4. An abnormally weak second sound at the pulmonary area and a distinct systolic murmur in early childhood are symptoms which can only be explained by congenital pulmonary stenosis, and are therefore not to be undervalued in differential diagnosis.

5. The absence of a palpable thrill, in spite of a very loud murmur, audible over the entire precordial region, occurs almost only with congenital abnormal openings in the septum and therefore points against an acquired heart affection.

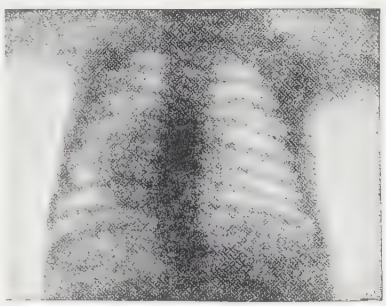
6. Loud, vibrating, systolic murmurs, with the point of maximum intensity in the upper third of the sternum, without symptoms of marked hypertrophy on the part of the left ventricle, are very important signs in

the diagnosis of persistent ductus Botalli; and they cannot be explained by endocarditis of the aortic valves.

Radiography of the Child's Heart.—While the radioscopic picture of the heart of older children shows no deviations from that of adults, the shadow of the heart in infancy and early childhood offers especial peculiarities on account of the proximity of the heart to the thymus gland and to the high position of the diaphragm.

In normal infants almost the same picture is seen in radiograms of the thorax, as well in adventral as in addorsal projection; flanked on either side by bright areas corresponding to the lungs, with shadows of the ribs passing horizontally across them, a central shadow is noted, narrow above, wide below, corresponding to the heart (*b*). The lower portion of the shadow spreads out more to the left than to the right; the left and right ventricles. The outline of the upper part runs parallel to that of the vertebral column or is somewhat concave, spreading a little to each side of the shadow of the vertebral column. This shadow which begins at the first or second dostal vertebra in the radiograms, follows the upper chest vertebræ and at the fourth, fifth, or sixth dorsal vertebra spreads out gradually, forming concave outlines asymmetrically downward, which surround the convex outlines of the ventricles of the heart. It must be noted that, in early childhood, the small upper portion of the central shadow must be due chiefly to the thymus gland, for the thymus, as is well known, surrounds the large vessels at their exit from the heart-sac, so that the large blood vessels can be imagined in the middle of this shadow. This differs from the radiogram of the adult thorax, in which the outlines of the upper portion of the central shadow are formed by the vena cava and pulmonary aorta. Besides, here also the large blood vessels cause but a small dark zone in the Röntgen picture, passing a little laterally beyond the shadow of the vertebral column, and spreading beyond the shadow of the vertebral column only when dilatation of the vessels occur.

Fig. 89.



Röntgen picture of the thorax of a perfectly normal, breast-fed child of three weeks, corresponding to the diagram in Fig. 88*a*.—The heart is normal and the thymus gland is of normal size, the shadows of which, united with that of the large vessels, are completely concealed by that of the vertebral column. Addorsal photograph, reduced to ⅓ in size.

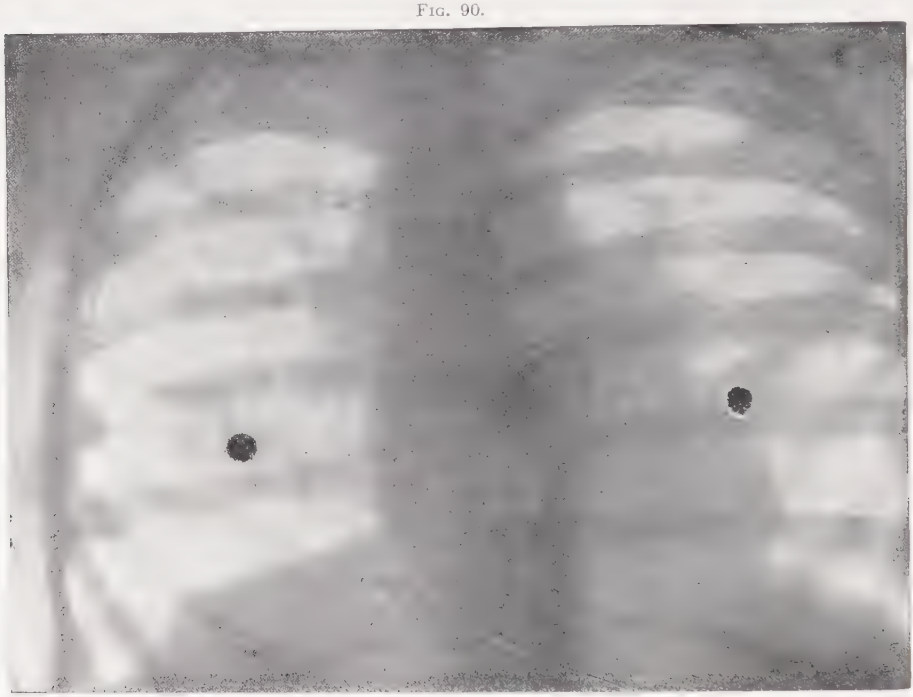
Röntgen examination of the normal infant's thorax shows a central shadow, in shape like a flask with a bulging body and narrow neck. The cervical portion of this flask-shaped central shadow extends from the first or second to the fifth or sixth dorsal vertebra. Its lateral outlines just approximate those of the shadows of the vertebræ (Fig. 89).

As we commonly find this picture in normal children entirely with-

out symptoms, in whom the dulness caused by the thymus gland does not extend beyond the edges of the sternum, it should be considered the normal shadow-picture of the young child, corresponding to both thymus gland and large vessels above, while the body of this flask-shaped shadow belongs to the mass of the heart.

By means of radioscopy the position of the apex, the size of the heart and its motility can be established in the shadows projected. By fixing the position of the nipples, the relations of the edges of the heart to the mammillary line are easily noted.

FIG. 90.



Normal heart of child ten years old.—The photograph is taken somewhat obliquely. The nipples are marked by white circles. The apex lies somewhat outside the mammillary line. The numerous dark, irregularly shaped spots, reaching from the cervical portion of the central shadow to the lung areas, are enlarged bronchial glands.

In order to prevent errors in estimating the relations of size and position of the pictures of the thorax in the Röntgen prints viewed, attention must be directed to the fact that only radiograms should be considered in which the tubes have been set up above the child in the median line, and at some distance from the child (in small children 50 cm. from focus to plate; in larger children, 60 to 80 cm., Kienböck).

Hochsinger's investigations have shown that exact estimations of the cardiac dulness have always agreed with the shadow of the heart in the Röntgen pictures. Radiography has proved invaluable in childhood for exactly estimating the position of the heart. Thus congenital median position of the heart is recognized at the first glance. The rela-

tions in the size of both sides of the heart are shown in an extraordinary manner. But radiography is most important in the diagnosis of dilatation of the pulmonary artery due to persistent ductus Botalli, of aneurysm of the aorta and pulmonary artery, and also in the diagnosis of pulmonary stenosis (see further on).

A means of differentiating whether the dilated pulmonary artery receives blood only from the right ventricle or also from a patulous ductus Botalli depends, according to de la Camp, on the nature of the pulsation of the shaded portion of the Röntgen picture. When a patulous ductus Botalli persists, a systolic pulsation, simultaneous with that of the arch of the aorta, but far more diffuse, occurs. As regards the shadows of the other blood vessels (the upper arches of the heart shadow, Fig. 90) it should be noted that information upon the position and size of the arch of the aorta, its regular origin and course, can be obtained; from dilatation and visible pulsation of the right upper shadow arch, dilatation or irregular origin of the aorta can be determined.

III. SPECIAL PATHOLOGY OF THE CIRCULATORY APPARATUS IN CHILDHOOD

1. PATHOLOGICAL PULSATIONS OF THE HEART

Irregularity of the pulse may occur in children as a physiologic or pathologic condition. In regard to the latter, the following forms of arrhythmia are to be differentiated:—that following intestinal disturbances and the actions of poisons, whether drugs or autointoxications; that seen in the convalescence from serious diseases; that found in diseases of the central nervous system; and that noted in organic heart diseases.

The normal relation between pulse-rate and frequency of respiration, as four is to one, is most important in childhood.

Continued retardation of the pulse-rate is always a pathologic symptom, and almost always means disease of the brain or chronic poisoning, endogenous or exogenous.

In the different cardiac affections of children the pulse varies in character. It is small and easily compressible in acute pericarditis and hydropericardium; almost always much accelerated and accompanied with galop-rhythm in acute myocarditis. In chronic myocarditis there are rapid pulse and arrhythmia. In dilatation of the heart the pulse is small and accelerated; with cardiac hypertrophy, it is full and accelerated. In acute endocarditis it is strong, full and rapid, sometimes irregular. Very rapid pulse (pulsus celer) does not occur in childhood. In later childhood it is always dependent upon excessive hypertrophy of the left side of the heart. Dicrotic pulse is also not observed in early childhood.

(*a*) TACHYCARDIA

The heart action of a child, always rapid, shows an acceleration under the most varied conditions. Fever, excitement, bodily overexertion cause a more rapid increase in the pulse frequency in childhood than in later life. The pulse beats regularly and the accelerated heart continues to beat with the same force, as long as the cause is operative. Paroxysmal tachycardia is different, in that very decided acceleration of the heart-beat occurs in sudden attacks, in conjunction with unpleasant sensations, palpitation, feeling of anxiety, outbreaks of perspiration, general weakness and exhaustion.

The dispute as to whether paroxysmal tachycardia is simply a neurosis or an acceleration of the heart-beat dependent upon an organic basis, has not been decided since Bouveret's fundamental work. The occurrence of this affection in childhood and its disappearance, without leaving a trace, during childhood or later in life, shows that this anomaly may certainly depend upon purely functional disturbances of the nervous system of the heart also. Only older children suffer from this condition. The duration of the attack varies between several hours and days, during which time the pulse-rate is rarely under 200.

The causes of paroxysmal tachycardia are in doubt, but in children especially, traumatic and psychic influences are considered the first occasions for the attacks, which sometimes alternate with other nervous symptoms, such as asthma, migraine, vertigo, fear of noises. In childhood especially, relations between paroxysmal tachycardia and clinical signs of cardiac affections are absolutely absent. Hochsinger knows of two cases of this condition in which attacks occurred regularly with high-grade constipation, and always ceased after thorough evacuation of the bowels. In the first child the attacks began in the eighth, in the second, in the tenth year. The latter recovered.

Symptomatology.—The attack may be of short or of long duration. Respiration is not accelerated during the attack, while the secretion of urine considerably increases, except in a case of Herringham's, in which it decreased. When the attacks are very frequent and the condition has lasted a long time, it may lead to cardiac hypertrophy, even in childhood, but this disappears with the disease.

Whether, in the cases in which there is no organic heart affection, the cause is paralysis of the vagus (Bouveret), stimulation of the accelerans (Tunker), or bulbar disturbance (Debove) remains undecided.

According to Hochsinger's observation, a third form of tachycardia occurs in childhood, perennial tachycardia, as the result of compression of the vagus by enlarged bronchial glands. Tachycardia may be the single clinical symptom of hyperplasia of the bronchial glands. Hochsinger has observed three conclusive cases in children from three to five

years of age, in whom permanent tachycardia occurred for about a year, following whooping-cough and rubeola, the pulse-rate remaining between 160 and 180; then it gradually ceased spontaneously. This could have been due to compression of the vagus only.

Treatment.—The treatment of paroxysmal tachycardia in children must regard the nervous origin of the condition and must prevent the reflex product of attacks. Stimulating drinks, copious meals and violent exertion are forbidden; the bowels must be kept regular. In some cases the paroxysm may be stopped by strong pressure in the epigastrium, by forcibly stopping respiration or by compression of the carotids until vertigo occurs. In severe attacks the application of cold to the cardiac region, or a spray of ether or ethyl chloride is of use. Drugs are useless during the attack. Narcotics, taken internally or applied externally, may lighten the subjective symptoms during the attack.

(*b*) BRADYCARDIA AND ARRHYTHMIA

Retardation of the heart action in childhood is almost always associated with irregularity of the pulse. Only this retardation of the pulse with arrhythmia in childhood is not always so marked, because of the higher pulse rate. Therefore periodic arrhythmia (double pulse) which is always caused by organic heart disease in adults, is hardly to be considered in children; much more frequently simple irregularity and inequality of the pulse are noted.

Physiologic arrhythmia, without retardation of the pulse, is an expression of the physiologic nervousness of children (Weill) which can be plainly observed, especially during sleep; but it may also occur from excitement, the irritation of cold or heat, or after unusual bodily exertion.

Arrhythmia is very rare in the heart affections of childhood, except in the diphtheritic heart, with which there is always high-grade bradycardia; and is more frequent in myocarditis and pericarditis than in endocarditis and valvular lesions.

More important is the arrhythmia accompanied by retardation of the pulse seen in the course of endogenous and exogenous poisons. All heart poisons may lead to this pulse disturbance, belladonna, digitalis, caffeine, opium, chloroform. Certain gastro-intestinal autointoxications, associated with constipation and acetonæmia, and jaundice may produce bradycardia and arrhythmia in children, while jaundice often causes an astonishingly low pulse-rate, 50 to 60 a minute.

In the acute infectious diseases it is not during the period of fever, but after the fever has fallen and during convalescence, that arrhythmia appears. As a rule, arrhythmia and bradycardia occur together, in the course of an infectious disease, at the lowest point in the temperature

curve, sometimes simulating a meningitic symptom-complex. The often high-grade bradycardia in diphtheria, in which the arrhythmia as a rule is less noticeable, depends upon toxic diseases of the heart muscle, as has already been mentioned.

Paroxysmal bradycardia, in a child 4 years of age, with a pulse of from 35 to 80, following inflammatory rheumatism and accompanying myocarditis, described by Schuster, is to be regarded as an unique observation.

Bradycardia and arrhythmia are often found associated in the course of organic diseases of the central nervous system. Tuberculous meningitis in children gives the lowest pulse-rate.

Simple arrhythmia, without bradycardia, is as a rule, found in chorea, in anæmic, nervous children, and in those suffering from intestinal worms. Arrhythmia is also frequently found with appendicitis in children and in acute intestinal affections with great loss of fluid. Attacks of migraine in school children are often accompanied by arrhythmia also, with constant, though slight retardation of the pulse.

Bradycardia, like tachycardia, can be produced by compression of the vagus with hyperplasia of the bronchial glands (von Starck), when the Stokes-Adams symptom-complex may appear, continued slow pulse with epileptiform and syncopal attacks (Charcot).

Pulsus paradoxus, described by Kussmaul, is not to be confounded with arrhythmia. Occurring with weakening of the radial pulse during inspiration, it is found in children with callous mediastino-pericarditis, with large mediastinal tumors, with the inspiratory spasm of laryngismus stridulus and in diphtheria (Variot).

While simple arrhythmia, associated with slight retardation of the pulse, is usually an ephemeral condition of little diagnostic importance in childhood, when it is accompanied by true bradycardia, it is almost always of longer duration and dependent upon deeper causes, such as severe disturbance in the action of the heart muscle, deeper changes in the nervous mechanism of the heart or organic changes in the central nervous system.

The **treatment** depends upon the nature of the fundamental disease.

2. CARDIAC HYPERTROPHY AND DILATATION WITHOUT VALVULAR LESION

Anatomy.—Slight grades of cardiac hypertrophy in children are only discovered with difficulty, even in examination of the cadaver, since the size, weight and thickness of the walls of the heart vary in the different years of life. Precise anatomic diagnoses are only obtained by weighing and measuring, and comparing results with the figures given for these relations by Müller, Beneke and Bizot (see page 441). As regards histology, attention should be paid to the size of the fibres of

the heart muscle, which are 4 or 5 times larger in adults, and to their greater slenderness in early childhood. E. Weill found a striking increase of the fibrillæ of the heart muscle with cardiac hypertrophy of renal origin in children.

As in adults, simple and eccentric, general and partial (right-sided or left-sided) hypertrophy are also differentiated in children. In left-sided hypertrophy the heart is enlarged downward and to the left, becoming cylindro-conic in form; in right-sided hypertrophy the heart forms the segment of a bow, due to increase in its horizontal diameter, with the string of the bow outlined by the left ventricle.

Cardiac hypertrophy as the result of angiosclerotic processes is rare in childhood and is always due to syphilis. As a rule hypertrophy and dilatation of the child's heart are conditions resulting from other diseases occurring inside or outside of the heart.

Etiology.—Congenital hypertrophy is infrequent without other cardiac diseases; in the first months of life acquired hypertrophy occurs, always associated with enlargement of the thymus gland, from Hochsinger's radiographic observations (Fig. 91). Though hypertrophy of the right side of the heart may exist for some time after birth, often as fœtal remains, hypertrophy of the left side of the heart, occurring between the third and fourth years of life, depends upon isthmus formation in the aorta which has not been completed early in all children (Gerhardt).

Eccentric hypertrophy of the heart may occur during whooping-cough and chronic bronchopneumonia; also with shrinking of the lungs and bronchiectasis. High-grade rickets may also have this effect on the heart, as the result of compression of the thorax and the pulmonary circulation. Very important among the causes of cardiac hypertrophy in children are renal affections. Deformities of the thorax (*kyphoscoliosis*), overexertion of the heart and the infectious diseases may also produce cardiac hypertrophy.

Germain Sée has considered an idiopathic hypertrophy of the heart due to growth at the age of puberty, which he explained as an independent overdevelopment of the heart as compared with the regular growth of the body. This view has been opposed by numerous writers, especially Potain and Ollivier.

According to E. Smith, moderate grades of cardiac dilatation occur very frequently in anæmic children who have grown rapidly, with chronic pulmonary affections, especially with bronchopneumonia. Hauser noted cor bovinum with enormous dilatation of the heart and signs of chronic congestion in a child of eleven months, who died of whooping-cough, without any change in the valvular apparatus.

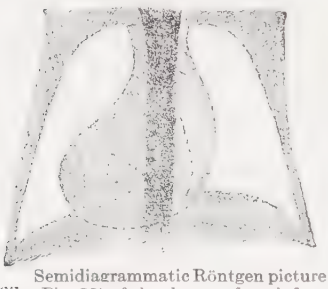
Alone stand the two cases of high-grade congenital idiopathic hypertrophy of the heart, described by Raissa Efron in infants of six

months and one year, produced by compression of the left bronchus. Autopsy in both cases showed a very large heart, hypertrophied equally in all its parts, without the slightest changes in the valves or ostia.

Symptoms.—A violent shaking in the precordial region, with forward arching of the precordium, downward displacement of the apex-beat, longitudinal increase in the cardiac dulness with hypertrophy of the left side of the heart, horizontal increase in the dulness when this hypertrophy is right-sided, and accelerated heart action are the essential symptoms of cardiac hypertrophy.

The cardiac impulse and the heart sounds are both weakened in dilatation of the heart. With dilatation of the right side of the heart are noted dilated veins, peripheral cyanosis and relative tricuspid insufficiency also; with left-sided dilatation, dilatation insufficiency of the mitral valve occurs, accompanied by a dull systolic murmur at the apex. Dilatation of the heart may be permanent or transitory, and is always a sign of weakness of the cardiac muscle. The appearance of dilatation of the heart in acute or chronic nephritis, whooping-cough or bronchopneumonia in children is always unfavorable.

Fig. 91.



Semidiagrammatic Röntgen picture (like Fig. 88) of the thorax of an infant of two months, with hyperplasia of the thymus and hypertrophy of the heart. The photograph is taken from behind. The cervical portion of the central shadow spreads to both sides, far beyond the shadow of the vertebral column. The heart itself is increased in both diameters. The outline of the normal heart and thymus gland is marked on the right side by a dotted line.

That simple cardiac hypertrophy may occur in childhood without other heart lesion must be considered settled. As the result of radioscopic investigations, Hochsinger found hyperplasia of the thymus gland regularly associated with cardiac hypertrophy (Fig. 91). The so-called thyroid heart is also found in later childhood, with all the symptoms so well known from the pathology of exophthalmic goitre in adults. The so-called eccentric hypertrophy of the heart, athlete's heart, is noted in children who began early to spend hours in arduous athletic exercises. Cardiac hypertrophy also occurs in children from frequent and violent nervous excitement. Masturbation certainly plays a prominent part in the production of this hypertrophy (masturbator's heart). Only exceptionally do both the last-mentioned forms of cardiac hypertrophy lead to insufficiency of the heart muscle in childhood; yet they may be given in later life as the cause of the early appearance of this unfavorable condition. Congenital narrowness of the arterial system (Virchow) can predispose to hypertrophy in early life by giving rise to insufficiency of the heart muscle.

It would be difficult to support the idea of hypertrophy of the heart in later childhood, due to growth, which, as was mentioned, has been advanced by Germain Sée. Much more probably this symptom-

complex, in which nervous conditions also play an important part beside the usual symptoms of cardiac hypertrophy, depends upon mechanical or nervous overstimulation of the heart, in which case the possibility of congenital narrowness of the arterial system should not be overlooked as the predisposing cause.

As the result of cardiac hypertrophy in the child a visible forward bulging of the anterior chest wall appears, together with a weakening of the respiratory murmur at the apex of the left lung, while gallop-rhythm and arterial vibrations are observed only exceptionally.

A. Neumann has described as dilatative heart weakness (coeur forcé) in childhood a symptom-complex which develops in anæmic children from dilatation of the heart, to which congenital debility and chronic nutritive disturbances predispose. The characteristic symptoms are palpitation, dyspnœa, general weakness after overexertion, displacement of the apex-beat, and horizontal increase in the heart dulness.

Diagnosis.—The differential diagnosis between pericardial exudate and cardiac dilatation may be difficult in early childhood. Retraction of the left lung or infiltration of its peripheral portions may also simulate cardiac hypertrophy. In the former case the pulsation of the pulmonary artery would be visible; in the latter, pathologic respiratory murmurs would appear; in both cases, in spite of the great increase in the dulness, the other symptoms of hypertrophy of the heart, having apex-beat and accelerated heart action, would be absent.

According to Blache and Sée physiological hypertrophy should be recognized by palpitation, dyspnœa, migraine attacks, displacement of the apex-beat to the sixth or seventh intercostal space, irregular pulse and a systolic murmur at the apex, which can always be extracardial only in simple cardiac hypertrophy and represents a cardiopulmonary murmur.

Prognosis.—The rapid and early appearance of cardiac hypertrophy, peculiar to childhood, acts as a protecting mechanism for the circulatory apparatus. This hypertrophy may completely disappear if the causes of it, obstacles to the circulation, overexertion or nervous excitement, are no longer present. Dilatation is always a sign of cardiac weakness (insufficiency of the heart muscle) and, after continued injury to the heart, may follow hypertrophy, on account of which the prognosis of simple cardiac hypertrophy, originally favorable, becomes unfavorable. Acute dilatation of the heart with nephritis and the toxi-infectious diseases of childhood is a serious disease with a very doubtful prognosis.

Treatment.—In simple hypertrophy of the child's heart, the cause of the condition should be treated, as the hypertrophy itself cannot be overcome. Subjective symptoms are obviated by the application of cold, rest of mind and body, and simple, non-irritating diet. Acute dilatation of the heart needs energetic treatment. Stimulants must be

given, such as camphor, caffeine and ether hypodermatically, and especially full doses of digitalis, if there is sufficient time (see later p. 517). Steffen advises extract of ergot in the treatment of acute dilatation of the heart in scarlet fever, as follows:

℞ Extract. ergot ..... 1.0 ..... gr. xv
Syrup. cinnamomi ..... 20.0 ..... f ʒ v
Aquæ destill ..... 80.0 ..... q.s. ad ʒ iii
M. Sig.—One teaspoonful every 2 hours.

Chronic dilatation of the heart is treated as described in the treatment of insufficiency of the cardiac musculature, on page 518.

Smith advises strychnine and iron together for dilatation of the heart in anæmic children and for the dilatation remaining after the infectious diseases. The most judicious preparation of strychnine for this is the tincture of nux vomica. Thyroid therapy is effective for the thyroid heart.

3. CONGENITAL ANOMALIES OF THE HEART

(*a*) IN GENERAL

In this section the cardiac anomalies which occur intra utero will be considered, whether due to congenital arrest of development or to fœtal endocarditis. The theory of the disturbances of development occurring in separate cases can only be touched upon briefly in this text book; those who wish further details are referred to the excellent works of Rokitansky, Heine, Peacock, Roger, Rauchfuss, Vierordt and to Hochsinger's treatise on "Congenital Heart Diseases" (Wiener Klinik, 1891).

It is also impossible, in this short article, to consider anatomically the numerous anomalies which occur and possibly belong here, or to tabulate them according to the history of their development; Hochsinger prefers only to discuss those congenital affections which are most important clinically.

Clinically four large groups may be separated:

1. Arrested development of the septa of the heart;

2. Stenoses of the arterial ostia in the heart and of the large arterial trunks;

3. Congenital anomalies of the large vessels (patulous ductus Botalli and transpositions of the vessels);

4. Congenital changes in the position of the heart.

Congenital heart lesions may be due to two kinds of causes, teratologic and inflammatory. Especial stress was laid on the former in Rokitansky's studies, perhaps too much at the cost of fœtal endocarditis. For example, he blamed anomalies of the ostia absolutely upon arrested development of the cardiac septa. Borsch and Lancereaux, on the con-

trary, have correctly called attention to the frequent intra-uterine inflammatory origin of stenoses of the ostia. Both causes act without doubt and they are very often found associated in a single case of malformation.

Buhl, Rauchfuss and Vierordt believe that the combination of stenoses of the ostia with considerable defect in the interventricular septum points to teratologic etiology, since the septum ventriculorum is completely formed by the end of the eighth week, and endocarditic processes are exceptionally rare before this time.

Some anomalies may occur isolated, as, for example, defects of the interventricular and interauricular septa, narrowness of the ostia and trunks of the pulmonary artery and aorta; transposition of the large arteries; direct communication between the large arteries as the result of incomplete development of the septum trunci arteriosi communis; patulous ductus Botalli, and anomalies in the shape and number of the semilunar and atrioventricular valves. Much more frequent, however, is a combination of different anomalies, in which no certain relation between the separate malformations in regard to origin can be recognized. The frequent occurrence of incomplete closure of the septa of the heart, with narrowed ostia, points to obstruction to this closure as the result of the stenosis. The common combination of pulmonary stenosis and persistent ductus Botalli is explained by the blood entering the aorta vicariously, being distributed through the branches of the pulmonary artery.

Pathogenetically, faulty development or faulty direction of the cardiac septa and of the primarily common vessel trunks plays the most important part, beside fœtal endocarditis, which is responsible for many cases of ostium stenosis; of the venous ostia at times also.

General Etiology.—The hereditary occurrence of congenital heart lesions in families is established without doubt (Strehler, Eger, Friedberg, Orth, Dobney, Arnone, Ferrannini, De la Camp). It is doubtful whether rheumatism and infectious diseases of the pregnant woman are concerned etiologically. Syphilis surely exerts some, though not a very conspicuous, influence (Gerhardt, Rauchfuss, Pott). Among 500 syphilitic children Hochsinger found a congenital heart lesion seven times. Eger established syphilis in the father three times, out of 12 children with congenital heart affections. Tuberculosis in the parent may be associated with congenital narrowness of the aortic system in the offspring; according to some authors, with the occurrence of pure mitral stenosis, also.

Blood relationship in the parents, traumata and psychic influences upon the mother during pregnancy are repeatedly brought forward etiologically.

The coincidence of congenital cardiac affections with other mal-

formations and with certain general disturbances of the nervous system (idiocy, deaf-mutism) is very important, since it leads to the conclusion that all injurious influences which cause psychic or mental degeneration in children may also be concerned in the occurrence of cardiac anomalies.

Among the somatic malformations the following are to be noted: harelip, polydactylism, syndactylism, abdominal fissure, transposition of the viscera, anomalous teeth, atresia of the anus and rectum, undescended testicles, horseshoe kidney, absent spleen, superfluous lobes to spleen, epispadias, hypospadias, uterus bicornis, cleft lip and palate, anomalous auricles to ear, thigh defects and many others. Hochsinger considers especially important the frequent occurrence of congenital heart lesions with cretinism and Mongolian idiocy, more frequently with the latter general somatic and psychic arrest of development than with the former.

General Symptomatology.—Congenital heart lesions, taken altogether, are often incorrectly termed "morbus Cœruleus," the blue disease of the French, since a large number of the more severe and more complicated congenital heart lesions may last an entire life time or for many years, totally without cyanosis; in fact a considerable number of cases are distinguished by a skin colored in exactly the opposite manner, a deep pallor, which, by French writers (Jules Simon) has been called, not very suitably, cyanose blanche or cyanosis alba.

The cyanosis in congenital heart lesions of children may be shown in various ways:

1. By continued intense blue coloring of the entire surface of the body, beginning at birth;

2. By partial blue coloring of the body, only affecting the peripheral portions of the body, beginning at birth;

3. By partial blue coloring as in 2, but occurring first later;

4. By general cyanosis as in 1, also first occurring later.

In these types cases with or without visible venous congestion are to be separated. The fact that the most high-grade cyanosis may occur in congenital heart lesions without dilatation of the veins of the neck shows that the cyanoderma of congenital cardiac lesions depends not so much upon congestion as on faulty admixture of the blood, since, as the result of abnormal circulatory communications and transpositions of the vessels, the systemic circulation is decidedly overloaded with venous blood from the very beginning. The frequent absence of cyanoderma, in spite of the existence of abnormal circulatory communications, is not incompatible with this explanation, since the so-called "mixed cyanosis" can appear only when there is a real excess of venous blood in the left ventricle or in the aorta. This is not the case in all abnormal congenital circulatory communications and depends, in the first place, upon whether the venous blood from the right side of the

heart flows chiefly into the pulmonary artery or not. In faulty origin of the aorta, from the right side of the heart, this cyanoderma shows its greatest development. It becomes greater with dilatation of the right auricle and stasis in the veins of the body. Then dilatation of the veins becomes visible, a rare phenomenon in the congenital heart lesions of early childhood. The eye-grounds also show tortuous and distended veins. With intense congenital cyanoderma, the color of the venous blood can be recognized in the retinal arteries. Neuroretinitis has been found by McHamill in a boy of nine years with congenital cyanoderma. Perhaps, too, an incomplete sprinkling of the lungs with oxygen-containing blood plays some part in congenital cyanoderma, as explained by Louis and Ferrus in 1823. The great frequency of pulmonary stenosis with congenital heart lesions need only be thought of.

According to Moussous, a congenital general hypoplasia of the pulmonary arterial system, analogous to the chlorotic hypoplasia of the aortic system, also plays a part sometimes in congenital cyanoderma.

The cyanosis may be very great right after birth, the skin of the children being plum-colored, darker than any cyanosis of later life. It may totally disappear again or fade considerably soon after birth. Or it may be altogether absent at birth in congenital heart lesions and first appear later, frequently during various diseases of the respiratory organs, and then remain constant. Cyanosis may also be intermittent, *i.e.*, only show after exciting conditions, energetic movements, forced expiration (congenital intermittent cyanosis of Variot). The children who at birth are deep blue in color do not as a rule live long. Children in whom cyanosis is slight soon after birth, but does not altogether disappear, commonly live months, sometimes years; while those without cyanosis at the beginning, or those in whom, when present at birth, it rapidly fades, may live a long life.

In congenital cyanosis, after it has lasted some time, structural changes are always found in the veins of the body, which are dilated and have hypertrophied muscle in their walls (Loubaud), which explains the great rarity of œdema in congenital cyanosis of children. The skin capillaries are dilated and tortuous, especially in the peripheral portions of the body.

The so-called clubbed fingers (Fig. 92) are closely related to the cyanosis of congenital heart lesions. This deformity of the fingers consists of a disproportionately stout, knobby swelling of the end phalanges, which, when it has lasted some time, is accompanied by enlargement and hour-glass-shaped curving of the nails, while the first and second phalanges are only slightly swollen and the rest of the hand is normal, except for a thickening about the heads of the metacarpal bones, which only appears after the condition has lasted several years. The toes show similar changes. When the cyanosis is of a very high grade and has

existed many years, spindle-shaped swellings of the knee- and elbow-joints appear, giving the picture of the "osteoarthropathie hypertrophiante" of P. Marie and E. Bamberger.

Anatomically the thickening of the joints of the long bones is as a rule due, without exception, to an ossifying periostitis, while the clubbing of the fingers and toes is usually dependent upon swelling of the soft parts only. The development of clubbed fingers is to be regarded as an early stage of hypertrophying osteoarthropathy; yet clubbed fingers can occur without the general bone disease mentioned, but not vice

Fig. 92.

Clubbing of the fingers in a five-year-old child.

versa. Chronic congestion and toxic action together lead to the bony deformity described. General osteoarthropathy is rare in childhood, but clubbed fingers are frequently found with congenital heart lesions and chronic pulmonary processes. They may be found with congenital cyanosis even in the second half of the first year of life.

Physical Signs.—The changes in percussion in congenital heart lesions are chiefly horizontal increase of dulness, the right ventricle and auricle being especially prone to enlargement (see p. 450 and Figs. 93 to 95).

The *auscultatory changes* in congenital heart lesions of children are the production of murmurs in the heart and anomalies in the intensity

of the second sound at the pulmonary cartilage. These murmurs vary very markedly in their intensity in childhood. Very loud murmurs, transmitted to a distance, or low murmurs, recognized only by an experienced examiner, are observed. Abnormally loud cardiac murmurs in infants and little children are an almost infallible sign of the congenital nature of the existing heart affection, a fact which deserves consideration. The timbre of the murmurs varies also in congenital changes from a soft, blowing murmur to a harsh, sawing, hissing, even roaring sound. Musical murmurs are also often found. If the latter, as well as all kinds of especially harsh murmurs, occur in infants and children of two or three years of age, they are of the greatest value in the differential diagnosis, if the question whether the cardiac affection be acquired or congenital has to be determined; and they are always a point in favor of the condition being congenital.

Blood Examination.—In 1889 Krehl recognized hyperglobulia as a characteristic appearance in congenital cyanosis. From the later works of Vaquez, Bahnholzer, Marie, Hayem, Vidal and Variot, it was settled that, beside hyperglobulia, there were in congenital cyanosis an increase in the size of the red blood corpuscles (up to 10 μ), an increase in the hæmoglobin and iron, disproportionate to the number of blood corpuscles, and an increase in the alkalinity and density of the blood (1070 to 1080). Hyperglobulia and macrocythæmia increase with the age of the patient (up to 8,000,000) and are as a rule noted but slightly in infants and young children. In an infant with intermittent cyanosis, Variot found intermittent hyperglobulia also (6,220,000 with the cyanosis, 3,960,000 in the interval between cyanosis). Hyperglobulia and macrocythæmia appear, as oxygen-carriers, to offer a functional counterbalance for the cyanosis (Fromherz). Calabrese considers the hyperglobulia to be due to irritation of the blood-producing organs by the overloading of the blood with carbonic acid gas.

A condition similar to the cyanoderma of congenital heart affections occurs in the permanent hyperglobulia with splenic enlargement and cyanosis, but without heart lesion, which has been described by Vaquez. As there are congenital heart lesions with very slightly pronounced physical signs, the differential diagnosis between these two conditions can only be made from the presence or absence of a disproportionately large spleen.

Respiration.—Dyspnœa occurs as a rule, with congenital heart lesions, though it is not always plainly recognizable when at rest. It always appears, however, with bodily exertion and is proportional to the grade of the cyanosis. With the cyanotic form of the congenital heart lesions of childhood, peculiar attacks of suffocation very frequently occur, which are often started by coughing or expiratory spasm and may be repeated several times a day. The children often lose con-

sciousness, as in epileptic attacks, become deep blue with much accelerated, thready pulse and if consciousness does not return, the child's face expresses the anguish of death. Such attacks may end fatally, although as a rule they last from 5 to 15 minutes, when the children regain consciousness. In adults and older children with congenital heart lesions, such attacks are rare, since such cases do not usually survive the first year of life. Only cyanotic children suffer from these attacks, children with congenital heart diseases without cyanosis never having them.

Growth and nutrition both suffer in children with congenital heart lesions. Delicate bony structure, the late development of sex, the scanty growth of hair, in a word infantilism occurs here as in congenital syphilis and tuberculosis. A predisposition to kyphosis certainly exists in children with congenital heart disease, independent of rachitis (Eger and Rauchfuss). Clubbing of the fingers occurs in three quarters of the cases, which is never congenital, but often develop in the first months of life (L. Fischer's case, reported by Schlossmann, in a child of 5 months).

The pulse in congenital heart lesions is accelerated at all periods of life, yet there are exceptions to this in older children (pulmonary stenosis with retardation of the pulse). The size and rate of the pulse depends upon the action of the left ventricle.

The temperature of the body is always subnormal in cyanotic children. Subjectively the patient feels this diminution in temperature as continued chilliness.

Since Rokitansky's teaching that patients with heart disease are immune to tuberculosis, many works on the occurrence of tuberculosis with congenital and acquired heart disease have appeared. Though they do not all agree with the teaching of Rokitansky, the slight predisposition of children with heart disease to tuberculosis is certainly established, with the exception of one anomaly, congenital pulmonary stenosis. While, according to Frommolt, the frequency of tuberculosis with valvular lesions of the heart, taken altogether, is just 8 per cent.; with pulmonary stenosis, according to Stoelker, it is 16 per cent., according to Rauchfuss, 14 per cent., according to Scheele and Gintrac, about 25 per cent., according to Vierordt, 28 per cent., and in the small statistics of Louis and Carriére, 43 per cent. and 50 per cent. Lebert lays great stress on the fact that at least one third of all those suffering from congenital pulmonary stenosis become tuberculous.

Prognosis and Course.—Certain forms of congenital cardiac anomalies give an especially unfavorable prognosis as regards duration of life. At the head of the list stands atresia of the aortic ostium, which permits of a life to be counted by days only. Somewhat better, but still always sufficiently unfavorable, are atresia of the pulmonary artery and transposition of the large vessels, with which survival over the first

two years of life rarely occurs. Complete absence of interventricular and interauricular septa (cor uniloculare) also gives an unfavorable prognosis, but pulmonary stenosis, if combined with defective septum or patulous ductus, sometimes permits of a long duration of life. Simple defects of the interventricular septum (Roger's disease) allow a long life.

In a series of children congenital heart murmurs spontaneously disappear, to return with other diseases of the respiratory organs sometimes; or they totally disappear, with the complete and permanent absence of subjective symptoms. Congenital affections of the heart with slight objective and subjective symptoms may give rise to serious disturbances after intercurrent diseases. Pulmonary tuberculosis (which frequently accompanies congenital pulmonary stenosis), pertussis, the different forms of pneumonia, severe rachitis and intestinal diseases have a deleterious influence upon children with congenital heart disease.

With the occurrence of convulsive attacks, of hæmorrhages into the skin, mucous membranes and lungs, the prognosis is the more unfavorable, the younger the child affected.

(*b*) SPECIAL PATHOLOGY OF CONGENITAL HEART ANOMALIES

1. *The Defects of the Cardiac Septa*

(*a*) Defects of the Interventricular Septum

A defective septum, permitting communications between the two ventricles, as well as a foramen ovale which remains open, is a very frequent congenital heart affection. It may occur as an isolated malformation, but is more often combined with stenosis of the ostia, especially with pulmonary stenosis.

Rauchfuss brought forward the idea, formerly generally believed but denied by Rokitansky, that septum defects are primarily due to arrested development of the membranous portion of the septum. The openings may be found either in the posterior septum or the anterior part of the anterior septum, which is formed before the membranous portion; or, finally, in the posterior part of the anterior septum, which is in place before the septum membranaceum. The openings may be so placed that they form an oblique canal in the cardiac septum or they may lead from one ventricle obliquely into the opposite auricle, or may put all four cardiac cavities into communication.

Endocarditis is very often associated with defects of the ventricles in extra-uterine life, about these apertures as well as on the valvular apparatus, with which the general symptoms of polyarthritis may be found. Anatomically the differentiation as to whether the changes at the ostium (affecting the mitral and tricuspid valves) occurred congenitally or only later is then impossible.

Only rarely is the interventricular septum totally absent (cor uniloculare). More frequently disturbances of development or the formation of perforations are found; sometimes the septum is reduced to a single membranous fold or a short projection, rising from the apex of the heart. Finally, there are also cases in which the septum has apparently developed completely, but shows perforations in one or more places.

Symptoms and Diagnosis.—In 1879 Roger described a symptom-complex in which a systolic murmur with its point of maximum intensity in the centre of the cardiac region (third intercostal space to the left) played the chief part, and he considered this murmur due to the presence of a defect formed in the interventricular septum. This murmur is harsh, decidedly high in pitch, heard over the entire anterior surface of the chest and also over the back, without being accompanied by a palpable thrill.

According to Roger the children affected suffer neither from dyspnœa, cyanosis, palpitation or especial acceleration of the pulse. The cause of this so-called *Roger's disease* is very frequently perforation of the interventricular septum. But many children with mitral insufficiency show the same symptoms, which makes the differential diagnosis between this and defective septum very difficult.

Defects of the interventricular septum may run their course without producing heart murmurs. The size of the defect has no effect on the absence or presence of murmurs. Small perforations may occur with murmurs and large defects without, or vice versa. The murmurs, in defective septum, arise as the result of the blood whirling about when both blood currents meet at the communicating opening in the septum, under the pressure of the contractions of the ventricles with each systole. The following conditions are necessary for the appearance of murmurs:

1. Difference in pressure between both ventricles.

2. That the perforation remain open during contraction of the ventricles.

Small and medium-sized perforations within the muscular septum can be closed by the contraction of the septum in systole. Condition 2 is wanting. With complete defect of the interventricular septum murmurs may be absent, because condition 1 is not fulfilled.

Incomplete closure of the interventricular septum is often combined with other cardiac anomalies, by which the symptom-complex established by Roger may be greatly modified.

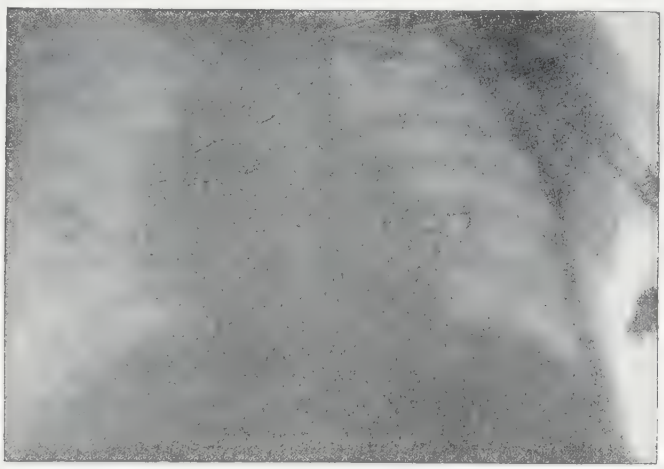
Accentuation of the second sound at the pulmonary area is important, a result of overloading the right side of the heart, to which blood is forced through the defective septum by the left ventricle, where the pressure is greater. With defect of the membranous (upper) portion of

the septum, this accentuation is most distinctly heard. Murmurs are never absent in this form of defect, so that we may correctly say, if septum defects produce murmurs, accentuation of the second sound at the pulmonary area is also present. The latter symptom almost always prevents mistaking the murmur due to septum defect for that of pulmonary stenosis.

In the diagnosis from chronic endocarditis of the mitral valve, stress is to be laid upon the absence or weakening of this systolic murmur at the apex, upon the audibility of the first heart sound under the murmur, and upon the slight increase in the size of the cardiac muscle.

If the child lives a long time, hypertrophy of the right side of the heart always results, which in pure cases always remains within moderate limits.

Fig. 93.



Röntgen photograph of a child 14 months old, with hypertrophy of both sides of the heart as the result of congenital defect of the interventricular septum.—The shadow of the heart, to the right especially, is shown extending far into the lung area. The cervical portion of the central shadow spreads equally to both sides of the shadow of the vertebral column, showing horizontal increase in the thymus gland.

More frequently than all other congenital heart anomalies, septum defects are associated with other disturbances of development, hypospadias, epispadias, cleft diaphragm, spina bifida, meningocele, etc. The lesions are well borne for a long time; death follows usually in later life, as the result of endocarditic valvular insufficiency. Sometimes a defective septum is a surprising accessory condition found at autopsy, even in old persons who have never complained of any cardiac symptoms.

(*b*) Defects of the Interauricular Septum

Patulous foramen ovale is frequently found at autopsy; according to Waldmann and Klob, in 44 per cent. of autopsies. More rare are perforations in the lower portion of the interauricular septum. Defects in the interauricular septum are only of consequence if, because of other

congenital or acquired heart affections, increased pressure occurs in the left ventricle, causing an overflow of blood into the right auricle and stasis in the veins of the body. So, too, with increased pressure in the right auricle, resulting from stenosis of the pulmonary artery or obstruction to respiration in the lungs, severe circulatory disturbances, with the appearance of cyanosis, may occur if the foramen ovale remains patulous.

Clinically a presystolic or diastolic murmur should be heard over the centre of the sternum, but this is seldom found with defects in the interauricular septum. Most cases run their course without anomalies in auscultation and the diagnosis is not made. In some cases systolic murmurs are also heard, but their relation to this anomaly is not at all determined.

The opinion of B. S. Schultze that the murmurs occurring here correspond to the diastole of the auricles is refuted by the production of pure systolic murmurs with defects of the interventricular septum.

Patulous foramen ovale is an anomaly the diagnosis of which can hardly ever be made, but it is found at autopsy as an accessory condition at all ages. The occurrence of this defect can be suspected in young persons, it relatively slight affections of the respiratory and circulatory apparatus rapidly lead to cyanosis.

2. *Stenoses of both Large Arterial Trunks*

(*a*) Stenoses of the Pulmonary Artery

In frequency and importance pulmonary stenosis ranks first among the congenital heart lesions. It forms three-fifths of all congenital anomalies of the heart and since this affection, associated with defective septum, allows longer duration of life than all the other congenital heart lesions, its frequency in later life is apparently even much greater than all the other congenital heart affections (four-fifths of the cases).

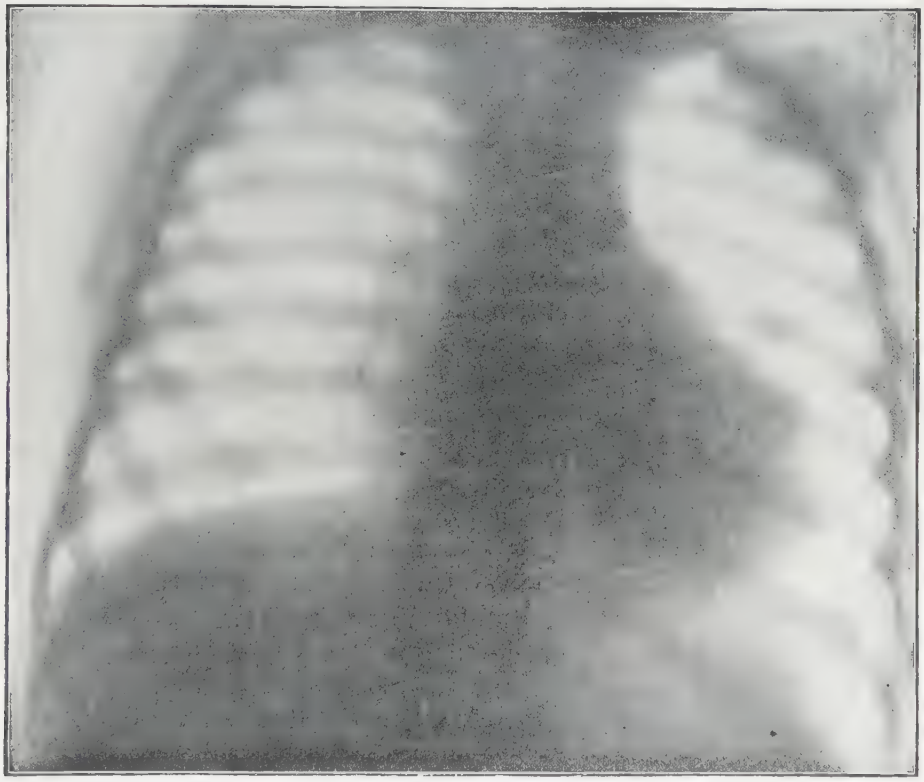
The stenosis may affect either the ostium or the vessel trunk, in its extracardial course, or at its intracardial origin (conus stenosis). Complete obliteration (atresia) of the pulmonary artery is very rare; on the contrary, pre-arterial or conus stenosis is frequent.

Atresia and stenosis of the pulmonary artery are the most common congenital heart lesions, Peacock found 112 (84.44 per cent.) out of 189 cases collected. Vierordt reckoned all the cases hitherto reported at about 300. Up to 1877 Asmus collected 47 cases of conus stenosis; in the past 20 years Vierordt collected 26, and recently Bennet, Buhl, Rauchfuss and Renvers have described new cases.

Sometimes the conus arteriosus becomes tied off at its origin from the ventricle as an accessory cardiac chamber, or there may be stenosis only at its apex, just below the ostium. The cause of stenosis of the

ostium may be fœtal endocarditis with adhesion, contraction and calcification, or arrest of development from anomalous division of the truncus communis (Rokitansky). Complete adhesion of the semilunar valves into a diaphragm which cannot oscillate has also been observed. Stenoses occurring early in fœtal life prevent the partition of the heart so that those portions of the septum which are formed under the aortic valves are usually absent. On this account the point of origin of the aorta is frequently shoved somewhat out of place. The foramen ovale

Fig. 94.



Röntgen photograph of a three-year-old child with congenital pulmonary stenosis.—The shadow of the heart is increased horizontally. The part of the shadow due to the pulmonary artery, between the second and third ribs on the left, shows slightly concave outlines, which may possibly be the result of atrophy of the trunk of the pulmonary artery.

almost always, and the ductus arteriosus very frequently, persist. In this case it is the duty of the latter to distribute blood from the aorta to the branches of the pulmonary artery. The scanty amount of blood carried to the lungs in high-grade stenoses is replaced by dilatation of the bronchial arteries vicariously; sometimes, also, of the œsophageal veins. The heart itself usually shows hypertrophy and dilatation of its right half. The action of the right ventricle may be arrested with complete atresia of the ostium.

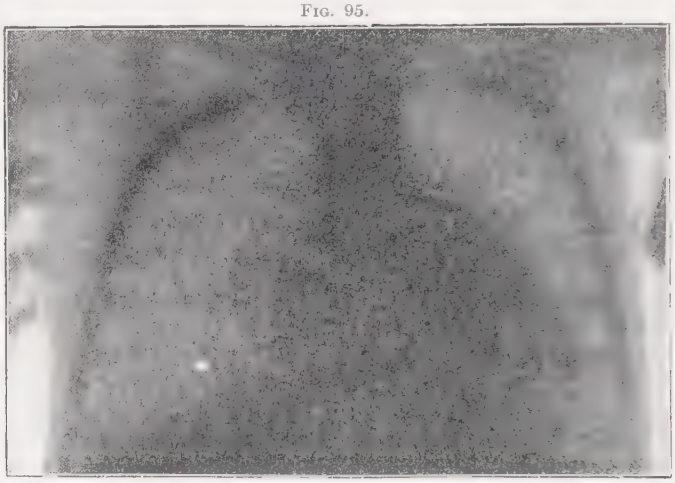
From the works of Kussmaul, Rauchfuss and Moussous regarding

the duration of life, Vierordt calculated an average life of from 12 to 13 years for pulmonary stenosis with simple complications. At least one-half of the children with pulmonary stenosis die before the tenth year. When combined with tuberculosis, the duration of life is apparently greater (about 18 years), an apparent contradiction, which is, however, explained by the fact that pulmonary stenosis is only complicated by tuberculosis later in life, at a time when most of the children with pulmonary stenosis have already died.

In a case described by Gutkind, a child with complete absence of the pulmonary artery lived for six years.

Symptomatology and Diagnosis.—*Physical Signs.*—The physical signs are increased cardiac dulness on percussion and abnormal auscul-

Fig. 95.



Röntgen photograph of the thorax of a child 4 years old, with eccentric hypertrophy of both sides of the heart, due to pulmonary stenosis with widely patulous interventricular septum.—In the third and fourth intercostal spaces on the left side the shadow of the heart spreads out very far, from which dilatation of the left auricle can be determined. The shadow of the blood vessels seems rather atrophied, as in Fig. 94.

tatory phenomena. Stress should be laid upon eccentric hypertrophy of the right side of the heart, which is as a rule but little pronounced in early childhood, since there exist, through the defective septum which is hardly ever absent, paths of outlet for the blood into the left half of the heart, which may at first direct both circulations. In older children the heart region is arched forward, the apex-beat made more diffuse and the cardiac dulness reaches disproportionately far beyond the right edge of the sternum.

Auscultation in most cases gives a systolic murmur over the ostium or the trunk of the pulmonary artery, with weakening of the second sound at the pulmonary area. For ostium stenosis, if it lasts a long time, is always accompanied by abnormal circulatory communications (defective septum, patulous ductus) so that the latter are to be considered in making the diagnosis.

The point of maximum intensity of the murmur is the left edge of the sternum between the insertions of the second and third costal cartilages, when the stenosis is combined with defective septum. Moving toward the right the murmur always grows rapidly less intense, but is loudest to the left of the sternum. As a rule the first heart sound at the pulmonary area is indistinctly audible or only incompletely suggested; only rarely does the murmur wholly take its place. Stenoses of the pulmonary artery have been described without murmurs and with cardiac hypertrophy and others with murmurs and without hypertrophy. Sometimes murmurs first appear near the end of life, without having been heard during many years; so, too, the disappearance of formerly perceptible murmurs has been observed. The absence of murmurs is explained in many cases, if the stenosis is high-grade, by the blood flowing into the left half of the heart through an open interventricular septum. When defective septum and pulmonary stenosis are combined, the murmur is often transmitted through the perforation in the septum to the aorta and into the carotids.

In childhood much more than in later life the observation of the quality of the second sound at the pulmonary area may lead to the correct diagnosis of pulmonary stenosis. With pure ostium stenosis of the pulmonary artery the second sound of the heart is always weakened, never accentuated, and if the children are small, it is never stronger than the first sound at the base of the heart. The smaller quantity of blood passing through the stenotic ostium of the pulmonary artery may cause, in diastole naturally, only a weak diastolic rebound against the semilunar valves, *i.e.*, a weak second sound at the pulmonary area. The simultaneous presence of a large defect in the interventricular septum can, after the child has lived a long time, exert an accentuating influence upon the second sound, since the left ventricle, under great pressure, aids in distributing blood to the pulmonary artery. Still, very marked accentuation is not to be expected from this combination. It is different with the simultaneous persistence of the ductus arteriosus, when the valves of the pulmonary artery have not suffered any damage in their ability to move and to oscillate. In later life, besides, as a result of the always increasing hypertrophy of the right side of the heart, an abnormally large quantity of blood may be driven into the pulmonary artery so that, contrary to all hypotheses, an accentuation of the second sound appears. Thus the quality of the second sound at the pulmonary area offers certain aid in diagnosis in early childhood only.

With pure conus stenosis also the second sound must be low. The second sound may be abnormally loud and may wholly obliterate the picture of pulmonary stenosis if conus stenosis is combined with patulous ductus or if ostium or conus stenosis is associated with dilatation of the pulmonary artery.

When the *diagnosis of congenital pulmonary stenosis* is made with certainty from the other symptoms, then with regard to the circumstances noted above, the following further rules of differential diagnosis may be deduced:

1. Abnormally weak second sound and absence of transmission into the veins of the neck lead to the presumption of pure stenoses or those combined with patulous foramen ovale only.

2. Clear but not accentuated second sound, with or without transmission of the murmurs into the veins of the neck, points to the simultaneous presence of an interventricular communication.

3. Marked accentuation of the second sound, with distinct transmission of murmurs into the carotids and subclavians, speaks almost with certainty for a patulous ductus arteriosus at the same time. This diagnosis becomes more certain if the murmur, as it reaches the neck, becomes vibrating in character, which is also observed in the veins of the neck, and if there are at the same time intimations of hypertrophy of the left side of the heart and palpable vibration of the arch of the aorta in the neck.

Subjective Symptoms.—The most prominent subjective symptoms are dyspnœa, attacks of suffocation, a tendency to fainting and vertigo. Very many of these children show the highest grade of cyanosis, which is increased to a very dark blue color by shrieking and crying, and by rapid movements of the body with the occurrence of the suffocative attacks. More than in the other congenital anomalies of the heart, all the causes leading to cyanosis (see p. 462) concur here. Very frequently such children are born apparently dead and deeply cyanotic, but recover. If the action of the left ventricle is sufficiently strong, considerable good health may exist for a long time, in spite of the cyanosis, although such children are easily chilled, stand mental and bodily exertion badly and are in general very susceptible. Many children with pulmonary stenosis die from pulmonary and exanthematous affections, but a large number of individuals live to reach later life, to 50 years or more. When loss of compensation appears, stasis results in the large viens of the body, with relative tricuspid insufficiency; the patients then die from dropsy.

In no congenital heart anomaly does clubbing of the fingers appear so early and so completely as in pulmonary stenosis.

(*b*) Congenital Stenosis of the Aorta

Congenital stenoses at the origin of the aorta are rarer than those at the beginning of the pulmonary artery and are divided anatomically like the latter into ostium and conus stenosis. Just as in pulmonary stenosis the valves may become adherent, forming a diaphragm with a central perforation, complete obliteration of the initial portion of the

aorta also occurs, in which case, if the child lives (*extra utero*), the pulmonary artery is dilated vicariously as a rule, the interventricular septum is widely patulous and the circulation of the body is kept up with difficulty through the open ductus.

A general narrowness of the aorta (hypoplasia of the aortic system) and congenital stenoses of this vessel beyond its origin are to be differentiated from the stenoses of the initial portion of the aorta.

Of especial importance is the so-called isthmus stenosis at the point of entrance of the ductus arteriosus. According to Theremin and Bonnet two anatomical types of isthmus stenosis must be differentiated. The first, which never leads to complete obliteration, is the result of arrested development of the isthmus aortæ, *i.e.*, of that portion which at an early period of fœtal life forms the connection between that part of the aorta which is to supply the upper half of the body and the descending aorta which branches off from the pulmonary artery. In this form the ductus Botalli is usually patulous. This stenosis affects that portion of the aortic arch between the left subclavian artery and the ductus Botalli.

The other, more frequent form is situated constantly opposite the insertion of the ductus ligamentum arteriosum and, as Skoda has taught, is due to contraction of that part of the wall of the arch of the aorta lying next to the ductus, since this portion must be supposed to be structurally like the wall of the ductus Botalli.

Congenital hypoplasia of the aortic system is characterized anatomically by abnormally thin walls, abnormal slenderness and dilatability of the aorta and its main branches. The aorta itself is frequently no wider than the normal carotid (see Fig. 101, *A*). The left ventricle is sometimes abnormally small, at other times considerably hypertrophied. The patients remain anæmic, small and weakly, and show delayed sexual development.

Symptomatology and Diagnosis.—1. *Congenital ostium stenosis of the aorta* cannot be differentiated from an acquired stenosis by the physical signs. Atresia of the mitral portion of the aorta, even when associated with patulous ductus, does not cause the production of murmurs. Murmurs may be absent with high-grade congenital ostium stenoses of the aorta, if the main blood stream is carried through a patulous septum to the right and onward through the pulmonary artery and ductus directly to the arch of the aorta. The length of life is limited in these cases, rarely extending over several weeks.

2. *Isthmus stenosis of the aorta* produces typical symptoms in later life, of which the most important is the development of a collateral circulation, the duty of which is to supply blood to organs receiving their blood from beyond the isthmus, chiefly those of the lower half of the body. The following arteries take part in forming this: anterior mammary, anterior intercostal, superior intercostal, dorsal and

transverse scapular, subscapular and external thoracic arteries, which carry the blood to the superior epigastric and posterior intercostal arteries. These arteries are seen and felt as tortuous pulsating or also vibrating, projecting cords, feeling solid just beneath the skin. The internal mammary arteries are most dilated and a systolic murmur may be audible in them.

The majority of the cases occurring in childhood run their course without the formation of a collateral circulation, if the stenosis is not of high-grade and the hypertrophy of the left ventricle, which is never absent, is still capable of overcoming the stenosis.

It is possible to make the diagnosis more frequently in children from the murmurs, which are always purely systolic, heard over the sternum, and to the right of it, in the upper intercostal spaces, up to

Fig. 96.

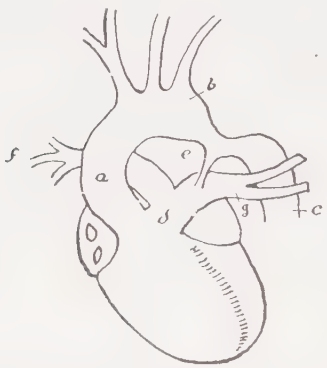


Fig. 97.

Diagrammatic representations of isthmus stenosis of the aorta. *a*.—ascending aorta; *b*.—isthmus aortæ; *c*.—descending aorta; *d*.—pulmonary artery; *e*.—ductus arteriosus; *f*. and *g*.—right and left branches of the pulmonary artery. In Fig. 96 the ductus Botalli has closed; in Fig. 97 it is patulous. (From Vierordt).

the neck; sometimes, if the murmurs have their points of maximum intensity upon the manubrium sterni, they are transmitted into the arteries of the neck, accompanied by a bulging forward of the arch of the aorta in the neck. In differentiating it from pulmonary stenosis, absence of weakening of the second sound at the ostium of the pulmonary artery is noted. An increased second sound at the aortic cartilage, with rapid, ringing rebound, if well marked, should be of great value in the diagnosis in children. Besides, retardation of the crural pulse and decided weakness of the pulse-wave in the arteries of the lower half of the body may be found very essential aids in support of the diagnosis, while the arteries of the neck and the arch of the aorta, which were still supplied with blood before the obstruction, are very full and dilated and show a bounding pulse.

The number of cases hitherto published is 90, according to Barie, of which by far the great majority occurred in males. According to Vierordt there are 69 males to 26 females with this affection.

These individuals may live a long time. Reynaud's case of a shoemaker aged 92 years, is well known. Vierordt estimated the average length of life for persons with isthmus stenosis to be 31.1 years for males, 30.3 years for females. The fatal termination of the disease occurs with symptoms common to cardiac disease, in which dyspnœa and dropsy play the principal part. Rupture of the aorta in front of the stenosed area, with sudden death, has repeatedly been seen. So, too, has rupture of the right auricle (Meckel) and of the right ventricle (Cooper) been observed with isthmus stenosis.

3. *Congenital narrowness of the aorta* and arteries of the body, with which Rokitansky was already familiar, has only been the object of

Fig. 98.

Cor bovinum in a child 11 months old, with transposition of the large vessels and rudimentary ascending aorta.—Photograph taken from behind. The right ventricle (RV) is enormously dilated and the dilatation is continued into the superior vena cava (Vcs).

comprehensive investigations since its relation to chlorosis was discovered by Virchow. In typical cases the heart remains abnormally small. But as a large number of individuals with this condition show signs of cardiac hypertrophy in childhood, it must be understood that the heart, too small at first, later hypertrophies at the time of puberty. Probably the symptom-complex described by Germain Sée as insufficiency of the child's heart due to growth (see p. 458) is nothing else than cardiac hypertrophy with congenital narrowness of the arteries of the body.

The clinical picture of this condition, in the investigation of which Bruberger, Leyden, Kulenkampf, Tuczek, Riegel, Küssner, and Ortner deserve much credit, is, taken altogether, that of a valvular lesion of the left side of the heart. The most frequent complaints are of palpitation, dyspnœa, and rapidly wearied mind and body.

The **physical signs** vary very greatly. If it is possible, radiographically, to show an abnormally small heart, with an abnormally slender shadow of the vessels, this is of incalculable value in the diagnosis of the condition. With hypertrophy of the heart the disproportion between the cardiac hypertrophy and the narrow carotid and radial arteries would be very real.

Murmurs may be present or absent in this congenital anomaly. They are easily understood, when the condition is complicated by a congenital heart lesion, but with a normal valvular apparatus they are dependent upon the autochthonous oscillations of the wall of the aorta, which is dilated with each systole of the heart.

Continued low temperature of the body and œdema have frequently been noted.

Vierordt collected altogether 30 cases which came to autopsy, 21 of which were in males, 9 in females, but only one of these cases affected a child. Symptoms of loss of compensation belong to the period of life after the development of puberty.

3. *Congenital Anomalies of the Large Vessels*

(*a*) Transposition of the Large Arteries

According to Vierordt about 70 anatomically authentic cases of this anomaly have been described up to this time, the pathogenesis of which depends upon anomalous partition and position of the primary truncus arteriosus, which has several variations, according to Rokitansky. The most important is true transposition (*transpositio vera*), in which the aorta occupies the place of the pulmonary artery and vice versa (complete inversion). The semilunar valves change their position with the transposition. The coronary arteries of the heart are then also abnormally placed. The transposed vessels may rise from one ventricle or the aorta arises from both ventricles, in equal or unequal parts, just over the interventricular septum, which is defective in such cases. The pulmonary artery may arise in the same way.

So, too, the openings of the veins into the auricles, like the large arteries, may have their positions changed, or the venæ cavæ and pulmonary veins may open into one auricle only, when fœtal communications will always persist between both circulations, in order to maintain the regular blood supply.

Defects in the septum usually exist with transposition of the arteries, somewhat more rarely the ductus Botalli persists, and sometimes, too, there are stenoses of the ostia of the heart.

When the aorta rises from the right side of the heart, its walls are thicker than those of the left side of the heart. In many cases the calibre of the transposed vessels is abnormal, especially the pulmonary

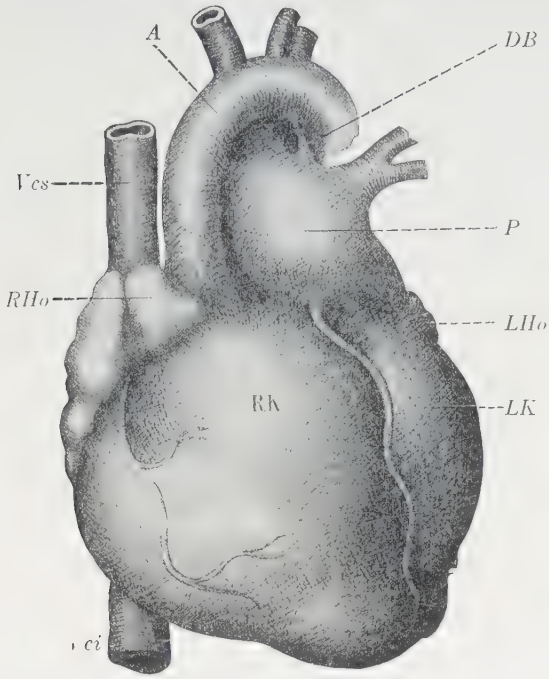


FIG. 99.—Congenital transposition of the large vessels with ectasis of the pulmonary artery and hypoplasia of aorta, in a child of five months.—Anterior view of unopened heart, natural size. *RK*.—right ventricle; *LK*.—left ventricle; *RHo*.—right auricle; *Vcs*.—superior vena cava; *Vci*.—inferior vena cava; *A*.—aorta; *P*.—pulmonary artery; *DB*.—ductus Botalli.

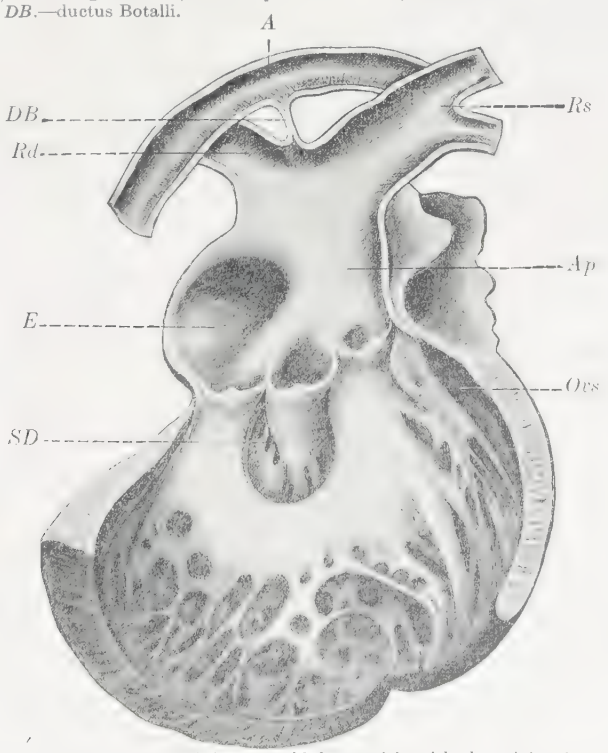


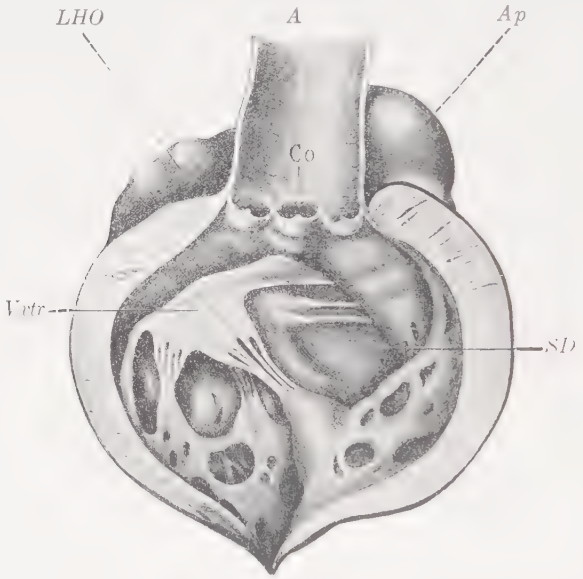
FIG. 100.—The same case.—View of the opened left ventricle with the origin of the pulmonary artery. *A*.—arch of aorta; *Ap*.—pulmonary artery; *E*.—ectasis of the pulmonary artery; *Rd*.—right branch of the pulmonary artery; *Rs*.—left branch of the pulmonary artery; *DB*.—ductus Botalli; *SD*.—defective septum; *Ovs*.—left venous ostium.

artery, when it rises from the left side of the heart, frequently appears abnormally wide; in one of Hochsinger's cases it was dilated as though by an aneurysm (Fig. 99).

With transposition of the large vessels, collateral communications between the capillaries of the branches of the bronchial and pulmonary arteries are very commonly found within the lungs, when the bronchial arteries are dilated and carry venous blood from the venous aorta into the lungs, which blood then, in a partially arterialized condition, flows back into the right auricle again, through the bronchial veins.

The length of life in cases of congenital transposition of the vessels is very limited, according to Taruffi and Vierordt. Most cases do not

FIG. 101.



The same case.—View of the opened right ventricle, with the origin of the abnormally small aorta. *A.*—aorta; *Co.*—coronary ostium; *LHO.*—left auricle; *Ap.*—pulmonary artery; *Vtr.*—tricuspid valves; *SD.*—defective septum.

survive the first six months. Of the 75 cases hitherto reported only 15 survived the second year of life. Exceptionally this anomaly is found between the twentieth and fortieth years.

Symptoms.—Sure symptoms of this condition can only be expected from pure transpositions. In complicated cases, associated with various other anomalies, obstacles to the diagnosis arise which cannot be overcome. Transposition of the origin of the arteries or at least a pulmonary artery arising from the left ventricle may be suspected if the second sound at the base of the heart is abnormally accentuated, without any murmurs being heard at the base of the heart, with pure heart sounds and with high-grade cyanosis. The abnormal accentuation of the second sound at the pulmonary area depends upon increased pressure within the pulmonary artery, supplied through the left ventricle,

which is also under high pressure. If, besides, a murmur is heard in the third intercostal space to the left of the sternum, there is probably at the same time a defect in the interventricular septum (see. Fig. 100, in which the diagnosis was made by Hochsinger).

Most cases of transposition are distinguished by high-grade cyanosis, yet sometimes cases have been observed with slight cyanosis, or without any cyanosis at all.

Suffocative and convulsive attacks are frequently found in older children with dark blue cyanosis. The heart is almost always hypertrophied, and sometimes it is changed in position also, having a median position or one to the right side.

(*b*) Persistent Patulous Ductus Arteriosus Botalli

Persistence of the ductus Botalli is only of clinical significance if it retains its original dimensions after birth, or increases in width in the course of life extra utero. Closure normally results from a growth of tissue which arises from the spindle-cells of the media of the vessel wall especially, with contraction of the other layers of the vessel wall,

Fig. 102.

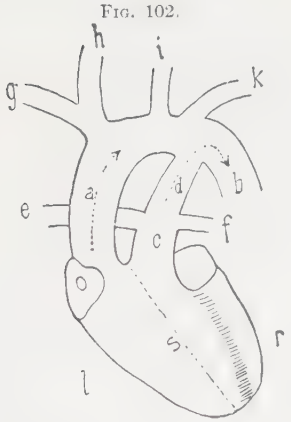
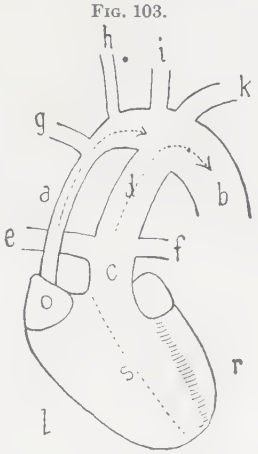


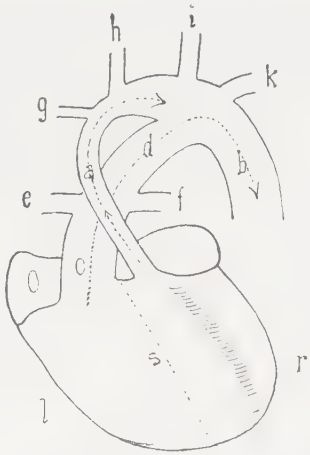
Diagram of simple patulous ductus Botalli.

Fig. 103.



Patulous ductus with rudimentary ascending aorta and large vessels in normal position.

Fig. 104.



Patulous ductus with rudimentary aorta and transposition of the large vessels.

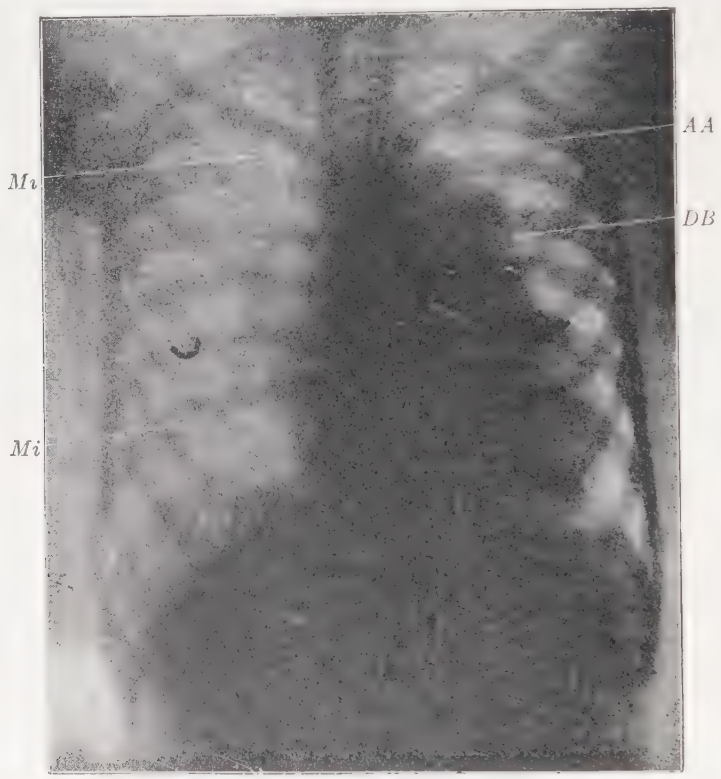
a.—ascending aorta; *b.*—descending aorta; *c.*—pulmonary artery; *d.*—ductus Botalli; *ef.*—branches of the pulmonary artery; *gh.*—right subclavian and carotid arteries; *ik.*—left subclavian and carotid arteries; *r.*—right; *l.*—left.

begins in the centre and is as a rule still not wholly perfect before the end of the first month of life. Incomplete closure occurs, in which the duct remains permeable to a sound during life, but this is of no significance. Absence of closure may depend upon abnormal nature of the wall of the duct or upon mechanical obstruction to obliteration, from imperfect respiration or disturbance in the pulmonary circulation during the first days of life. In this relation congenital cardiac anomalies

are the first causes, followed secondly by atelectatic conditions of the parenchyma of the lung. The patulous ductus may either be of equal width throughout its course or it may dilate like a funnel toward the aorta, or finally it may be shortened so that aorta and pulmonary artery communicate directly through a common opening. Aneurysmal dilatation has been observed with disease of the wall of the duct.

The first clinical studies upon the isolated occurrence of patulous ductus Botalli were those of Almagro and Duroziez (1862) in which

Fig. 105.



Patulous ductus Botalli with aneurysmal dilatation of the ductus and pulmonary artery in a boy of thirteen. *AA.*—arch of the aorta; *DB.*—ductus Botalli and pulmonary artery dilated like an aneurysm, giving a cap-shaped top to the shadow of the heart. *Mi.*—internal mammary artery, considerably dilated, denoting an internal collateral circulation.

attention was called to the occurrence of murmurs of a peculiar sort, which may be absent in the first weeks of life but usually develop within the first six months, with increasing dilatation of the duct.

The mechanical results of patulous ductus are increased pressure in the pulmonary artery and an increased flow of blood from the aorta into it, which in time leads to dilatation and inflammatory disease of the wall. So, too, hypertrophy and dilatation of the right ventricle must eventually result.

Patulous ductus may exist alone or in association with other con-

genital anomalies. Up to this time about 25 uncomplicated cases of this condition have been described anatomically.

The diagnosis of patulous ductus was evolved by François Franck and Gerhardt.

With any considerable dilatation of the ductus, *percussion* shows a band of dulness in the first two intercostal spaces, projecting for the width of a finger over the left edge of the sternum, corresponding to the dilated pulmonary artery (Gerhardt's band of dulness), which corresponds in Röntgen pictures to a peculiar cap-shaped top above the shadow of the heart (see Fig. 105).

For the occurrence of murmurs with patulous ductus the following conditions are necessary: (1) sufficient width of lumen; (2) a sufficient flow of blood under sufficient pressure, from the aorta on one side and the pulmonary artery on the other, since the murmurs in question depend upon the formation of eddies by the unequal blood current within the combined vessels, ductus and pulmonary artery. As a result, then, in a ductus with small lumen, or when a patulous ductus is combined with high-grade stenosis, atresia or arrested development of the aorta or pulmonary artery, murmurs may be absent. The murmurs, almost always purely systolic in the first years of life, may be accompanied by diastolic murmurs in later years, as a result of inflammatory diseases of the walls of the duct and of the pulmonary artery. The murmurs are very well transmitted to the back; François Franck wrongly considered this a specific symptom of patulous ductus.

Auscultation gives either pure heart sounds or systolic, more rarely also diastolic murmurs with their points of maximum intensity on the manubrium sterni, well transmitted to the neck and back.

The behavior of the second sound at the aortic cartilage is of great importance in diagnosis, as it is always very considerably accentuated in pure cases and frequently produces a palpable shaking of the chest wall with diastole (palpable rebound in the second intercostal space on the left side).

The murmur is transmitted into the carotids, more to the left than the right. With any considerable dilatation of the ductus and sufficient distribution of blood through the pulmonary artery, the vibrating arch of the aorta is palpable in the neck. With dilatation of the pulmonary artery at the same time a systolic vibration is felt in the three upper intercostal spaces to the left of the sternum, to which a strong palpable rebound is added in diastole.

François Franck laid stress upon the occurrence of pulsus paradoxus (diminution in pulse-rate with inspiration and increase with expiration) in the diagnosis of patulous ductus, yet this symptom is not in the least constant.

Just as inconstant, too, is the symptom recently made prominent

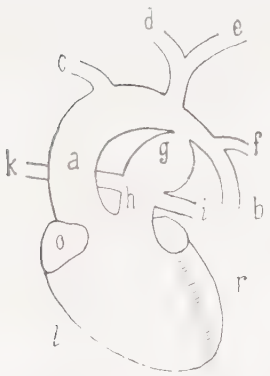
by Dokuszajewa, of a decreased pulse-wave in the left half of the body, which would be well founded, theoretically, if the ductus were widely patulous and the blood stream of the aorta were decidedly diverted.

In the pure forms of patulous ductus Botalli, cyanosis is almost always absent. Subjective symptoms first develop as a rule at the end of the first year or even later. Of these the most important are palpitation, dyspnœa and a tendency to pulmonary catarrh. This cardiac anomaly sometimes allows of long life, yet only a few cases have lived past 40 years.

The difficulty in **diagnosis** is great when patulous ductus is associated with pulmonary stenosis, especially if the valves of the pulmonary artery have lost their ability to oscillate, since the important symptom of accentuation of the second sound in the pulmonary area may then be absent. The differential diagnosis from simple pulmonary stenosis is made from the palpable rebound of the second sound, from the transmission of the murmur and the palpable vibration in the neck, and, not least, from the Röntgen pictures which show an increase in the shadow of the vessels of the left side in patulous ductus, but in pulmonary stenosis an atrophied or normally sized shadow (see Fig. 95).

The free communication between aorta and pulmonary artery as the result of embryonal defect of the septum trunci, a great rarity, gives no certain symptoms of clinical value.

Fig. 106.



Diagrammatic representation of dilatation of the ductus Botalli and pulmonary artery. *a.*—ascending aorta; *b.*—descending aorta; *c.*—right subclavian artery; *de.*—both carotids, rising from a common trunk; *f.*—left subclavian artery; *g.* aneurysm of the ductus Botalli and pulmonary artery, *h.*—pulmonary artery; *ik.* right and left branches of pulmonary artery; *r.*—right; *l.*—left.

In older children rupture of the ductus may occur (Röder, Esser). Typically the site of the rupture is near the point of entrance of the ductus into the pulmonary artery. Before rupture occurs, a dissecting aneurysm of the ductus and terminal portion of the pulmonary artery sometimes develops.

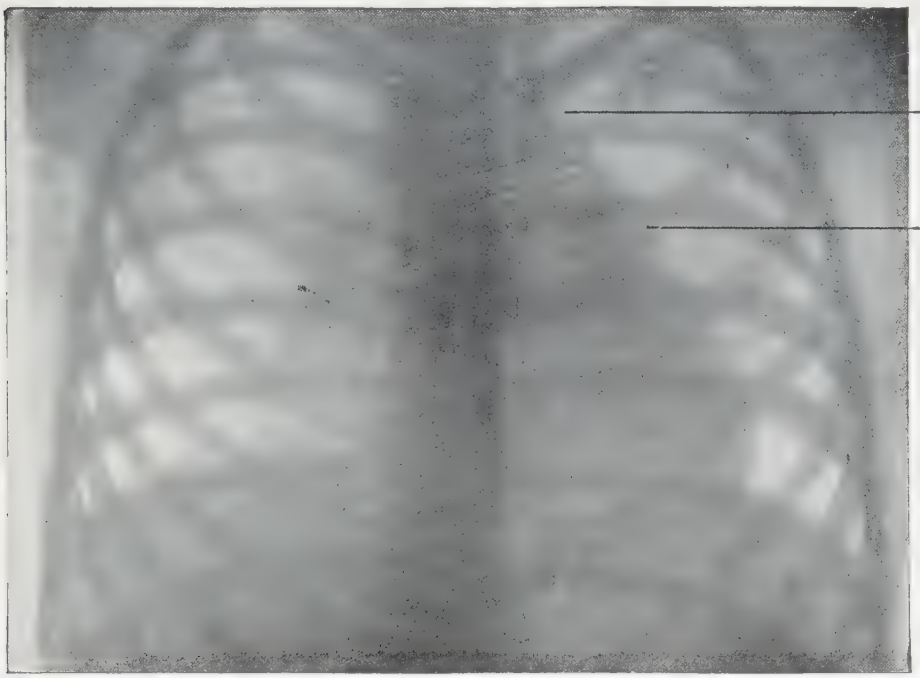
DILATATION OF THE PULMONARY ARTERY

In recent years cases of aneurysmic dilatation of the pulmonary artery with and without patulous ductus Botalli have been frequently described, by Dresler, Arnheim, Krzyszkowski and Wiczkowski, Weinberger, Burke. Without discussing the question whether in every case these were aneurysms in the anatomic sense of the word or simply dilatations of the initial portion of the artery, the fact is important that congenital ostium stenoses, of the pulmonary artery especially, may be associated with cylindrical or sac-like dilatations of the vessel behind the ostium, which are dependent upon congenital weakness and delicacy

of the arterial wall. These dilatations are as well recognized in Röntgen pictures as those associated with patulous ductus (Fig. 106). Theoretically, as regards the differential diagnosis in such cases, the shadow of the pulmonary artery should be more sharply defined from that of the aorta than when a patulous ductus is also present.

The diagnosis of simple dilatation with ostium stenosis, remarkable to relate, has never been made during life, but in all these cases the condition was supposed to have been patulous ductus Botalli.

FIG. 107.



Röntgen photograph of the thorax of a child of three years, with aneurysmic dilatation of the pulmonary artery.—As no oscillation was palpable in the neck, the ductus Botalli is probably closed.—Aneurysm of the pulmonary artery.

Proportional dilatation of the pulmonary artery with patulous ductus, giving a systolic blowing murmur, with its point of maximum intensity over the pulmonary area, with accentuated second sound, was described by Zuber in a child of five months.

CONGENITAL CHANGES IN THE ATRIOVENTRICULAR OSTIA

These anomalies form a very small portion of the congenital heart affections. The venous valvular apparatus, of the right side of the heart especially, is affected very frequently, whether in the form of arrested development up to the complete absence of ostium and valves, or in the form of fœtal inflammatory processes and their results, stenosis and insufficiency. Indeed, congenital tricuspid and mitral insufficiency

and stenosis are not rare, though they never occur as isolated affections, but always in association with other anomalies, the foremost of which is defective septum. The diagnosis of the isolated affection does not differ from that of the same cardiac anomaly when acquired. When in combination with other congenital cardiac changes, the diagnosis of the limits of the changes in the venous ostia of the heart is scarcely to be thought of.

Complete absence of the tricuspid valve, in association with pulmonary stenosis, making the right venous ostium insufficient, has recently been described by Spolverini and Barbieri, in an apparently healthy boy eleven years old.

The same writers have observed atresia of the mitral valve in a cyanotic boy with a systolic murmur but without other anomalies, who died when forty days of age.

4. *Congenital Changes in the Position of the Heart*

Dextrocardia, with or without transposition of the cardiac cavities (complete or incomplete) is either a partial symptom of complete situs inversus or it arises as an isolated displacement of the organ and is then almost always found to be associated with other errors of cardiac development.

Mesocardia (median position of the heart) is synonymous with a preservation of the position of the fœtal heart in the centre of the thorax. This abnormal position is not so rare (Filatów) and the diagnosis is to be made if the apex-beat is felt in the epigastrium, the heart sounds are heard clearly behind the sternum and no adhesive changes between the heart and neighboring organs can be found.

The congenital changes in the position of the heart, whether accompanied by situs inversus or not, are almost always associated with intracardial arrest of development.

Ectopia of the heart is of more anatomical than clinical importance. With ectopia the sternum is wholly or partially absent and the skin may be normal or may also be lacking. Ectopia may be an isolated arrest of development or may be but part of an eventration, in which the greater portion of the anterior chest and abdominal wall is absent, so that the viscera of the thorax and abdomen come to lie outside of the body. Both ectopia, in which the heart may be covered by pericardium or be absolutely uncovered, and eventration do not allow of living.

For all other teratologic anomalies of the heart reference should be made to the text books upon pathologic anatomy.

Finally, primary congenital ptosis of the heart, discovered by Rummo, which Ferrannini, who found it with ptosis of the viscera, considers as a partial symptom of a general ptosis, should not be passed

without mention. Ptosis of the heart occurs together with low position of the diaphragm. Ferrannini found congenital (?) mitral stenosis at the same time in four cases.

CONGENITAL ANOMALIES IN THE SIZE OF THE HEART

The thickness of the wall and capacity of the separate portions of the heart undergo changes in utero, with the various congenital anomalies. In stenoses of the pulmonary artery, hypertrophy and dilatation of the right side of the heart occurs; so, too, the left side of the heart is affected by congenital stenoses of the aorta. This retrodilatation and hypertrophy increases after birth. Rules are of no more value here than elsewhere in cardiac pathology. Only by abnormal circulatory communications can the affected portion of the heart disburden itself so that an isolated increase in one part of the heart is sometimes absent, and the entire heart muscle becomes hypertrophic (cor bovinum congenitum, Fig. 98). As a rule, with atresia of the arterial ostia, the half of the heart on that side atrophies, while the other side of the heart hypertrophies. Besides, there are enormous congenital hypertrophies of the heart, the cause of which is not always wholly visible (Hauser, Hueter). The highest grades of congenital atrophy of the heart are found with obliteration of the atrioventricular ostia.

THE TREATMENT OF CONGENITAL HEART LESIONS

An effective treatment of congenital heart lesions does not exist. In general the measures which are of value soon after birth in congenitally cyanotic children, to keep them alive, differ from those which are used in later life. Breast-feeding, protection against loss of body temperature (by incubators at first), and the prevention of infections are urgently necessary during the first weeks and months of life. As all diseases of the respiratory tract have unfavorable effects on children with congenital heart diseases, one's chief aim is toward preventing them. In older children the easy methods of "hardening" are valuable (fresh air, cold baths and massage). Sojourn in a southern climate during the winter will best prevent catarrhal affections of the upper air-passages.

The treatment with drugs begins in children who have attacks of asphyxia, with symptoms of insufficiency of the heart muscle. Convulsive attacks are best combated by the regular administration of large doses of bromide, 0.5 Gm. (7½ gr.) daily to infants, 1 to 4 Gm. (15–60 gr.) for older children. During fainting spells hypodermatic injections of ether, caffeine sodiobenzoate or sodiosalicylate or camphorated oil are used. For the cyanosis frequent inhalations of oxygen are advised, though its success is doubtful.

4. DISEASES OF THE PERICARDIUM

(*a*) PERICARDITIS. INFLAMMATION OF THE PERICARDIUM

Etiology and Occurrence.—Although circumscribed areas of cloudiness and thickening of the pericardium in the region of the apex and the points of entrance of the venæ cavæ are frequently found at autopsy in the hearts of children, pericarditis in childhood is in general less common than in the later years of life, doubtless because of the very much greater rarity of rheumatism in childhood. Therefore the recognition of pericarditis in the living child offers greater difficulties than in adults, and frequently enough, especially in infancy, it appears as a surprise at autopsy. According to the statistics of Cnopf and Weill pericarditis is only one half as frequent between the second and fifteenth years as in later periods of life, and is most frequent in childhood from the seventh to the tenth year. Pericarditis is most rare from the second to the seventh year of age, and somewhat more frequent in the first year, on account of the pyæmic infections which enter more into the question etiologically at that time of life.

Cnopf found pericarditis seven times (5.38 per cent.) out of 130 autopsies performed upon children. The most frequent causes of the occurrence of pericarditis in children are the acute infections, especially the exanthematous diseases, scarlet fever, measles, varicella and erysipelas; it may also result from inflammation of the neighboring organs (lungs, pleura, mediastinal glands, thymus gland, œsophagus and bones of the chest wall). Acute and chronic nephritis are also frequently associated with pericarditis, not to mention articular rheumatism, which enters less etiologically into the question of pericarditis in early childhood, because it occurs more rarely at this period than in later life. So, too, the infection of the inflammatory diseases of the abdominal cavity sometimes spreads directly to the pericardium in children.

Banti found, in two cases of pericarditis associated with pneumonia, Fränkel's diplococcus in one and in the other the staphylococcus aureus and albus. In a child operated upon by Körte the pericardial exudate contained staphylocci, streptococci and short bacilli. In another case examined by Banti, pericarditis with nephritis, the exudate was sterile. Pneumococcus infection was primary in a case described by Coutts, without pneumonia.

The occurrence of an idiopathic pericarditis is questionable. Unnoticed rheumatism or traumatism play a part in cases in which the etiology is obscure. Naturally tuberculosis is also an etiologic factor in the occurrence of pericarditis in childhood and may spread especially easily from diseased mediastinal glands.

It is a peculiarity of childhood that pericarditis in the earliest periods of life depends chiefly upon pyæmic infection; in middle child-

hood, mainly upon the spread of inflammatory processes; and in later childhood, generally upon rheumatic, and also choreic foundation. Corresponding to this, the exudate in early life is usually purulent, in middle childhood generally serofibrinous, and in later childhood almost always purely fibrinous.

An especial peculiarity of childhood is the exceptionally frequent combination of pericarditis with endocarditis, which occurs when the condition is due to rheumatism and chorea. Weill and Henoch lay stress upon the possibility, in childhood especially, of the appearance of an endopericarditis before the general symptoms of rheumatism. Not rarely to a condition which at the beginning only affected the endocardium, pericarditis may later be added, with equal frequency whether the cardiac disease was at first acquired or congenital.

Pathologic Anatomy.—Anatomically there are only slight differences between pericarditis in children and adults. Perhaps purulent exudates are relatively more frequent in young children than later. Pericarditis at an early age more frequently leads to obliteration of the pericardium. More rarely does the process go on to deep-seated disease of the cardiac muscle, such as purulent infiltration and progressive fatty degeneration. The very intense injection of the blood vessels and the rapid appearance of abundant liquid effusion are characteristic of acute pericarditis in childhood. Purulent pericardial effusions are as a rule metastatic processes, the result of puerperal sepsis or osteomyelitis, or they occur from the spread of purulent inflammation of the lungs, pleura or mediastinal glands. Hæmorrhagic effusions are found in childhood in Werlhof's disease, infantile scurvy (Seidlitz and Kyber) and in tuberculosis. Besides, as in adults, acute and chronic, dry and exudative pericarditis must be differentiated. Dry pericarditis is commonly found as an accessory condition with endocarditis in children.

Symptoms.—The symptoms depend essentially upon the form of the pericarditis. In dry pericarditis the most important symptom is a friction murmur which can frequently be felt. The friction murmur may vary in character. Sometimes it is a low touching or rubbing sound, at others a stronger, grating sound, which is heard irregularly above the heart sounds and is continued longer than these. This murmur is usually loudest at the base of the heart and changes in intensity in children with change in position or pressure with the stethoscope. It becomes louder when the child sits up, walks or bends over or when pressure is made with the stethoscope; and lower when the child lies on its back. It is more easily heard in children than in adults, but the differential diagnosis between endocardial and pericardial murmurs is sometimes very difficult, in children especially. This is true when valvular affections exist with pericardial affections, when both kinds of murmurs come into question. If the pericarditis spreads, a second

point of maximum intensity of the friction murmur is as a rule noticed, at the apex. Friction murmurs are sometimes heard also in forms of pericarditis with effusion, in certain positions of the body, varying with the cases, in which both layers of pericardium, covered with fibrinous exudate come near enough to touch with the beats of the heart. As pericardial layers are kept separated with larger effusions, the friction murmur usually disappears when the exudate grows larger, and in its place appears a decided enlargment of dulness, which increases gradually as the apex-beat grows weaker.

Small effusions in children produce no change in the cardiac dulness and often no other symptoms of importance. Larger effusions give that triangular area of dulness, well known in the symptomatology of pericarditis in adults, of which the apex lies at the third or second rib according to the amount of the effusion, while the left side of the triangle—and this is especially characteristic—reaches far to the left of the point where the apex-beat is palpable.

Very frequently with large effusions, especially in small children, a general diffuse vibration or undulation of the entire anterior chest wall is palpable, with weakened or absent apex-beat, while a marked forward bulging is never absent.

Large effusions produce visible dilatation of the left half of the chest which does not move to and fro with respiration. The heart sounds are weak, pendulum-like, embryocardial in type. The pulse-rate is considerably accelerated, sometimes reaching 150 to 180 to the minute in small children.

The apex-beat, in exudative pericarditis of children, may move to various places within the area of dulness, by change of the child's position, if the heart has not lost its motility in the pericardium filled with effusion. When the child bends over or is placed in the knee-elbow position, the apex-beat, which may previously have been absent, may reappear, an important sign of exudative pericarditis.

Pericarditis of children escapes recognition very frequently when associated with left-sided pleural effusion. A massive pericardial effusion, too, considerably compressing the left lung, might be mistaken for a pleural effusion if sufficient stress were not laid upon the position of the apex-beat of the heart which is shoved to the right side with pleurisy, and is absent with pericarditis.

The **general symptoms** of acute pericarditis are not characteristic at the beginning. Palpitation, rise in body temperature, sometimes arrhythmia and pain in the precordial region point to a cardiac affection. The friction murmur or triangular dulness first make the diagnosis certain. In the chronic forms, subjective symptoms are sometimes very slight and are totally concealed by the symptoms of the fundamental disease. Important is the rapid appearance of high-grade

dyspnœa in small children, even with slight effusion, with which cyanosis may for a long time be absent. Decided orthopnœa with increase in the cardiac dulness and weak apex-beat must lead to the diagnosis of pericarditis in children, even without the presence of the friction murmur. Tuberculous pericarditis often runs its course entirely without symptoms.

Symptoms due to changes in the heart muscle are never lacking if the pericarditis lasts some time. Acute dry pericarditis may rapidly lead to insufficiency and dilatation of the cardiac musculature, especially in delicate children; the exudative form, by compression, may produce serious circulatory disturbances. The addition of pericarditis to heart lesions in children exerts an unfavorable influence upon the compensatory relations of the cardiac muscle. As the result of large pericardial effusions, compression of the left lower lobe of the lung occurs very frequently in children. Pins' *pseudopleuritic symptom* of pericarditis depends upon the disappearance of the symptoms of pleurisy (dulness, friction murmur and diminished vocal fremitus) when the patient is placed in the knee-elbow position. According to Guinon this change in physical signs does not occur at once when the position is changed, but only after some moments. According to Aviragnet and Olsnitz, Pins' symptom can be found in exudative pericarditis in childhood.

Prognosis and Course.—The course of pericarditis is different whether it be dry or exudative in form, and the latter varies according to the kind of effusion. But pericarditis belongs to the most serious diseases of childhood, as complete recovery seldom occurs, and even in cases running a favorable course, adhesions between the layers of the pericardium may result, which will be decidedly deleterious to later life, and to further bodily development. The best prognosis can be given in pericarditis due to acute rheumatism which if there be no cardiac lesion, may recover without any evil result. Pericarditis following pneumonia and the acute infectious diseases is less favorable, chiefly because the resistance of the heart has always been decreased previously by the toxins of the disease. Purulent and hæmorrhagic pericardial effusions, with sepsis and tuberculosis, naturally give an absolutely fatal prognosis. If death occurs, it always is dependent upon insufficiency of the heart muscle and œdema of the lungs.

While circumscribed inflammations of the pericardium, especially those which occur with heart lesions in children, may appear and disappear without symptoms, other forms, with recurrent severe attacks of articular rheumatism, run a characteristic course, with eccentric hypertrophy of the heart which develops rapidly, asystolia, marked pain, great pallor and stenocardial attacks. Death from pericarditis in childhood rarely occurs with the appearance of dropsy.

Pericarditis is the most serious complication of rheumatism in childhood. Cadet de Gassicourt rightly says that all children who are killed by rheumatism die from pericarditis. While the mortality rate of rheumatism in adults is 3 to 4 per cent. (Besnier), this reaches 6 per cent. in children (Cadet de Gassicourt), and only on account of its being complicated by pericarditis.

Diagnosis.—The diagnosis of pericarditis in children has been covered essentially in the presentation of the symptomatology of this disease. Only the differential diagnosis between pericardial effusion and acute dilatation of the heart requires some words, especially as both conditions occur frequently with the same disease, scarlet fever. In both conditions the cardiac dulness is increased, the apex-beat weakened or absent, the heart sounds dull and faint. Here Pins' symptom and the return, with change of position, of the apex-beat which had disappeared, may sometimes decide in favor of pericarditis; yet it must not be forgotten that the last-mentioned sign may be absent in pericarditis, if adhesions have formed between the pericardium and the heart.

It is not, as a rule, difficult to recognize the nature of the exudate. The simultaneous occurrence of pyæmic symptoms points to a purulent effusion, while the exudate after rheumatism is usually serofibrinous.

The pericardial friction murmur, at the beginning of the disease in children, is very commonly purely systolic. In early childhood it is often so soft that it is easily mistaken for an endocardial murmur; yet it is not transmitted beyond the precordial region and its point of maximum intensity is not at the apex, as in acute mitral endocarditis of children.

In small children with slight purulent pericardial effusion, physical signs may be wholly absent. Sometimes the presence of most severe cardiac weakness which cannot be explained by the other symptoms may lead to the probable diagnosis of purulent pericarditis.

Treatment.—A prophylactic and causal treatment of pericarditis in children may be mentioned. The physician should always give his full attention to rheumatic symptoms in childhood and should never be satisfied with the inconsiderate diagnosis of "growing pains" when older children complain of pain in the joints and muscles. In the majority of these cases rheumatism is masked and the treatment with salicylates may offer protection to the heart. The same thing is true of choreic and scarlatinal pains in the limbs of children.

Whether the treatment with salicylates should be continued when rheumatic pericarditis has developed depends upon the state of the cardiac muscle. Dry pericarditis should always be treated with salicylic preparations at the beginning. To small children one or two grams of sodium salicylate, aspirin, salipyrin or salophen are given daily; double

this dose to older children. Inunctions of the precordial region with salicylic preparations, such as mesotan or rheumasan are of value when the stomach will not tolerate salicylates internally.

An unconditional therapeutic requisite is absolute rest, with the application of cold to the precordial region (best done by using small tubes through which cold water runs). Symptoms of cardiac weakness must be treated according to the rules given on page 517.

In chronic effusions the external application of preparations of iodine (tincture of iodine, iodvasogen) may be tried. Mercurial ointment may be employed interchangeably with preparations of iodine. Very large exudates, which permit of no other treatment, must be evacuated by surgical intervention; here it should be noted that puncture is as a rule an inadequate procedure, while incision, laying the pericardium widely open, with resection of the ribs and establishing drainage but avoiding irrigations, fulfils all indications.

Puncture, which, performed with caution, should be an exploratory puncture with a hypodermic syringe, is to be done in the fifth or sixth intercostal space. It sometimes saves life (cases of Biedert and Jürgensen) but it in no way hinders the reaccumulation of pericardial effusion. Incision has frequently lead to permanently successful results in children (Rosenstein, West, Dickinson, Sievers) and is as a rule performed in the sixth intercostal space near the sternum.

(*b*) PERICARDIAL ADHESION

Etiology and Occurrence.—Adhesion of the pericardium with the heart is more frequent in childhood than is generally supposed and is as a rule a condition resulting from plastic rheumatic pericarditis. Not rarely the pericardial adhesion develops slowly, wholly without symptoms, in older children with rheumatic polyarthritis; or it follows a valvular lesion of the heart without having previously caused the characteristic symptom-complex of pericarditis, and by its appearance makes compensation decidedly more difficult. In general the causes of pericardial adhesion are the same as those of pericarditis, rheumatism, chorea, scarlet fever and tuberculosis. It should be noted that symphysis pericardii is found much more frequently at autopsy in children than is recent pericarditis itself. Very rarely does purulent pericarditis lead to adhesion of the layers of the pericardium.

So far as age is concerned, obliteration of the pericardium has been found by Billard and Hüter in newborn infants; by Bednar in a child of three months; Morel-Lavallée found it eighteen times in children out of 30 cases. Among 43 cases which came to autopsy Cerf found three in the first decade of life and fifteen in the second decade.

Pathologic Anatomy.—The pericardial cavity is abolished and the heart is covered with a thick fibrous mass, which may reach 2 cm.

in thickness, even in small children. Just as in later life, so, too, in small children, adhesions of the outer surfaces of the pericardium may form with the sternum, ribs, lungs and diaphragm, results of an external pericarditis, which is combined with internal pericarditis (mediastinopericarditis) as frequently as it occurs as an independent disease of the mediastinal cellular tissue. When the disease exists a long time, deposits of lime and of cartilage are found between the heart and neighboring organs in childhood. In fresher cases the intermediate layer joining the external and internal layers of the pericardium is still soft, fibrinous and easily separated. Tuberculous adhesions may be cartilaginous lumps or semifluid, salty or caseous masses of exudate.

In early childhood the heart is almost always decidedly hypertrophied with symphysis pericardii; when this has lasted some time, there is high-grade dilatation, frequently causing relative insufficiency of the arterial and venous valvular apparatus. Secondary fatty degeneration of the myocardium is rare in childhood. The functional disturbance due to pericardial adhesion is very intense in children and is due to the arrest of growth of the child's heart primarily, depending upon obliteration of the pericardium.

Symptoms.—Taken altogether, two main groups of pericardial adhesions may be differentiated clinically: those which are associated with marked cardiac hypertrophy, usually of rheumatic origin; and those in which the heart only increases slightly in size, chiefly due to tuberculosis.

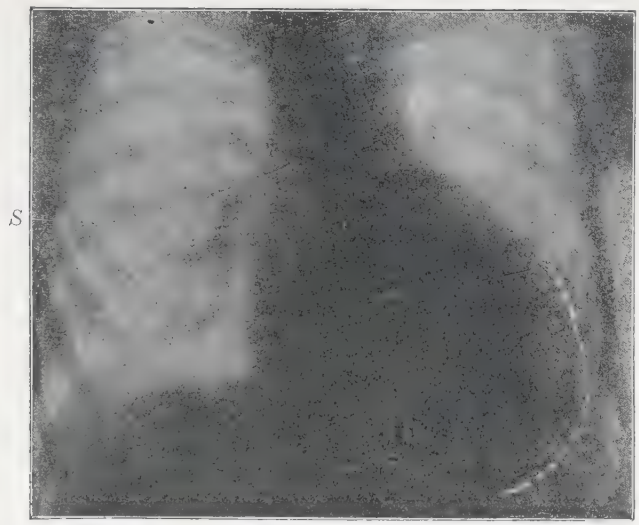
Whether the heart is hypertrophied or small, the most important physical signs are a systolic in-drawing of the chest wall over the entire precordial region and the absence of motility of the heart dulness with respiration and change of position. This systolic in-drawing of the thorax wall is of decided value if all the intercostal spaces entering into question are moved at the same time, especially if the region of the apex-beat and the base of the heart, as well as the xyphoid process, are all drawn in simultaneously with systole. This is observed with tuberculous obliteration chiefly, in which thick, lumpy adhesions between the pericardium and the chest wall occur. Under these conditions Röntgen photographs may help in making the diagnosis, in which the immobility of the heart and the extrapericardial bands and adhesions can sometimes be recognized (see Fig. 108, Hochsinger's case).

A certain intensity of the cardiac power and the presence of adhesions between the outer layer of the pericardium and its surroundings (chest wall, diaphragm, mediastinum) are necessary for the appearance of this systolic in-drawing. The tuberculous forms show this condition more frequently than obliteration of the pericardium due to other causes.

Auscultation of the heart gives but few certain signs in support of the diagnosis. Weill observed fœtal rhythm of the heart sounds many

times. Reduplication and splitting of the heart sounds, disappearance of the diastolic sound and a metallic character of the first sound have also been observed in pericardial adhesion in childhood. The last-mentioned symptom depends upon adhesion of the apex of the heart with the diaphragm and modification of the first heart sound from the vibration of air in the gastric cavity. With incomplete adhesion a pericardial friction murmur may also be heard, besides. As a result of dilatation insufficiency of the mitral and aortic valves, systolic and diastolic murmurs may appear (Schöneich), which are of no decided value in diagnosis since they may be caused by the valvular lesions of endocarditis existing at the same time.

Fig. 108.



Displaced cor bovinum in a boy of nine years.—Photograph taken from in front. The heart is shoved to the left side, placed horizontally, and fixed by adhesions in this faulty position. The pericardium is obliterated and adherent to the chest wall. The shadow of the right half of the thorax is darker as the expression of an obsolete right-sided pleurisy. At *S* are mediastinal callosities.

Decided value in the diagnosis, however, in childhood especially, is attached to the functional disturbances caused by pericardial adhesions. The tendency of the heart to contract and to grow is hindered by the obliteration of the pericardium. This leads to symptoms of pulse weakness and cardiac insufficiency which increase with advancing age. In conformity with this, pericardial adhesion frequently causes death, even in childhood. Indeed, it can be said that most of the causes of death from the acquired heart affections of children are due to the additional occurrences of pericardial adhesion. The most severe congenital and acquired heart lesions, even when several conditions are associated, may be well borne for years during childhood, thanks to the extraordinary compensatory ability of the musculature of the child's heart; but as soon as obliteration of the pericardium is added,—and this some-

times develops wholly latently,—symptoms of congestion and œdema appear which lead to death.

Tuberculous adhesions of the pericardium sometimes run the course apparently of an increasing dilatation of the heart, characterized by small pulse, weak apex-beat, palpitation and œdema. When these symptoms have lasted a long time, if tuberculous processes are discoverable on the other serous membranes, the differential diagnosis of tuberculous symphysis pericardii is made possible, as opposed to dilatation of the heart. Sudden death from cardiac insufficiency has been seen in older children with this condition (Schöneich's case, a boy of 8 years).

Pericardial adhesion is relatively more frequent in childhood than in adults. According to Marfan, the two chief forms of this condition, rheumatic and tuberculous, are differentiated from the contrasting relation of the volume of the heart which is decidedly increased in the former and small in the latter. Rheumatic symphysis pericardii is as a rule associated with valvular changes, while the tuberculous form is not. Both forms end fatally with symptoms of myasthenia. The investigations of Hutinel, F. Pick and Moizard have made clear the intimate relation between hyperplasia of the liver and obliteration of the pericardium, and the twofold reaction of the liver in the forms of perihepatitis and a sort of cirrhosis has been shown. In the tuberculous form especially, in which cardiac symptoms are frequently totally absent, a condition arises which might easily be mistaken for tuberculous peritonitis or alcoholic cirrhosis of the liver.

Diagnosis.—The diagnosis of adhesion of the pericardium is only made with certainty if that portion of the heart lying further to the left side is drawn inward regularly with each systole. The diagnosis is the more certain if the heart is not excessively large, in which case a systolic sinking-in of the intercostal spaces is not rare, even though the force exerted in this in-sinking is decidedly less than that noted when the whole chest wall is drawn in with adherent pericardium. Almost always, too, the in-drawing of pericardial obliteration is followed by a diastolic rebound of the parts of the chest wall previously drawn in (seen even more plainly in children than in adults), a thing which never occurs with simple cardiac hypertrophy.

The appearance of pulsus (disappearance of the radial pulse during inspiration) and inspiratory distention of the veins of the neck (Kussmaul) are uncertain symptoms which depend upon compression of the large blood vessels by mediastinal callosities. The same thing is true of Friedreich's diastolic collapse of the jugular veins which, as is well known, also occurs with tricuspid insufficiency and widely open foramen ovale.

In later childhood obliterating pericarditis is sometimes one of the symptoms of chronic sclerotic, often tuberculous processes, of the various serous membranes, in which the peritoneum, especially that

portion covering the liver, is most frequently affected (sugar-crusted liver). When this is associated with symphysis pericardii, ascites may appear early, a condition which is sometimes of diagnostic value in the recognition of this general disease of the serous membranes.

Treatment.—When the adhesion has reached the fibrous stage all treatment is useless. Whether separation of the pericardial adhesions surgically, as has already been attempted in later life, will prove of value in childhood remains undecided. In early life especially, when arrested cardiac growth is most important, freeing the heart of its constricting bands and callosities would be doubly valuable. In the rheumatic form of pericardial obliteration, as long as attacks of rheumatism still occur, the administration of salicylates is indicated, as in pericarditis. The treatment of the symptoms of insufficiency of the cardiac musculature is given on page 517.

5. ACQUIRED AFFECTIONS OF THE ENDOCARDIUM

(*a*) ENDOCARDITIS IN GENERAL

Etiology and Occurrence.—In its etiology, occurrence, symptomatology and course, endocarditis in childhood differs considerably from endocarditis in adults. It is found as a congenital condition, with or without arrested cardiac development, and attacks especially the valvular apparatus of the right side of the heart, and the arterial valvular apparatus rather more than the venous. On the contrary, in extra-uterine life the atrioventricular valvular apparatus on the left side of the heart is chiefly attacked. Fœtal as well as extra-uterine endocarditis of childhood usually seeks for its seat the valves which are exposed to the greatest tension. Fœtal endocarditis has already been treated among the congenital heart lesions. Here Hochsinger briefly considers as endocarditis that acquired after birth.

As regards the frequency of endocarditis in general, the figures covering a period of ten years, from the Children's Dispensary in Florence, are of value. It was found in 67 out of 4948 children, affecting the mitral valve in 54 (in six pericarditis and aortic affections were present at the same time), while in one case there was pure aortic endocarditis.

Rheumatism plays the principal part in the production of endocarditis in childhood. From Weill's large statistics, 60 per cent. of the endocarditis of children was found to be rheumatic in nature. Church found the endocardium affected by rheumatism in 80 per cent. of children with endocarditis. With this understood, stress should be laid on the fact that, in comparison with the slight frequency of rheumatism among children, endocarditis in childhood cannot be called a rare disease. According to Hochsinger's investigations, acquired endocarditis is very rare before the fifth year; then it rapidly increases in frequency and is most frequent between the tenth and fourteenth years. Both sexes are equally affected.

Endocarditis in children is only rarely idiopathic, being as a rule a secondary disease. Among Weill's 258 children with endocarditis,

150 were due to rheumatism,
39 were due to chorea,
15 were due to tuberculosis,
12 were due to scarlet fever,
7 were due to measles,
7 were due to diphtheria
7 were due to pneumonia
4 were due to typhoid fever
3 were due to dysentery,
2 were due to erythema nodosum,
and 12 were due to unknown causes (idiopathic endocarditis).

To the causes given in Weill's statistics smallpox, chicken-pox, relapsing fever, pyæmic and septic processes and erysipelas should be added. Syphilitic endocarditis also occurs in children.

With regard to the rheumatic etiology, cases published as occurring in infancy in association with endocarditis must remain in doubt. Joints may be swollen in infancy with endocarditis, but they are always dependent upon gonorrhœa, syphilis or pyæmia, not upon rheumatism. The youngest child suffering with rheumatic polyarthritis whom Hochsinger has observed was two and one half years of age. In the rheumatic etiology of endocarditis of children, besides, nodular rheumatism, chronic arthritis of children and acute and chronic muscular rheumatism (*torticollis*) are to be mentioned. In children more than in adults is it true that the endocardium acts like a synovial membrane. Not rarely endocarditis first appears after the second or third attack of rheumatism. Church found endocarditis with the first attack of rheumatism in 57 per cent. of children; with the second attack, in 75 per cent. West found endocarditis in 62 per cent., Cheadle in 80 per cent., Weill in 60 per cent., Fuller, in 54 per cent., Cassel in 62 per cent. and Hochsinger in 57 per cent. of the cases of children with rheumatism.

Concetti has recently taken quite a different stand, denying the intimate relation between rheumatism and the child's heart, as he found slight endocarditis only once among eight cases of acute rheumatism. On the whole he found disease of the left side of the heart in connection with rheumatism in 27.65 per cent.; 19.15 per cent. were due to acute infections and in 53.19 per cent. none of the etiological conditions mentioned could be found.

Concetti supposes a congenital cardiac dystrophy as the predisposing cause of the cases not founded upon infectious diseases.

Affections closely related to rheumatism, purpura and erythema multiforme, also bear an etiologic relation to the endocarditis of children. Rheumatic iritis has been seen combined with endocarditis, as with chorea also (Hohlfeld, Forster).

Of the other fundamental diseases to which endocarditis in childhood may be due, chorea comes first, causing 15 per cent. according to Weill, 13 per cent. according to Bonnaud, and 15 per cent. according to Hochsinger, of the cases of endocarditis in children. From Hochsinger's

experience, about one half of all children with chorea develop endocarditis, some with, others without symptoms of rheumatism, a condition which supports the opinion, advanced at present by Cheadle and Heubner, that chorea is a rheumatic equivalent. Cassel who does not consider this relation between rheumatism and chorea established, found rheumatism fifteen times among 38 children with chorea; and rheumatism, chorea and endocarditis together nine times. In no other disease, besides, is the differential diagnosis between organic and accidental heart murmurs so difficult as in chorea minor.

According to Weill a frequent anatomic finding with general tuberculosis of childhood, especially with tuberculous meningitis, is tuberculous endocarditis of the valves of the heart, which, however, does not lead to functional disturbances.

Among the exanthematous infectious diseases, scarlet fever stands first in causing endocarditis, yet the figures vary with the nature of the epidemic. Complete recovery occurs from the endocarditis of scarlet fever especially frequently. Pneumonia, epidemic influenza and endemic "grippe" should also be mentioned as more frequent causes of endocarditis. Endocarditis following measles and gonorrhœa is a great rarity in childhood.

Idiopathic endocarditis is not acknowledged by many writers (Cheadle, von Dusch) who believe these cases to be dependent upon rheumatism which has been overlooked, or to be primary rheumatism of the endocardium. But the exclusive appearance of cases of idiopathic endocarditis in children during the first four years of life, when rheumatism occurs uncommonly rarely, is hardly in favor of the above-mentioned view. From Hochsinger's observations, he considers the occurrence of an idiopathic endocarditis, comparable to fœtal endocarditis, as absolutely established in early childhood.

So, too, the occurrence of a purely traumatic endocarditis in children cannot be denied (cases of Prandi in a boy of 8, and of Kantorowitz, in a boy of 11 years).

According to Vianello and Cacchiole, ordinary infections of the nasal, pharyngeal and laryngeal mucous membrane play a part which should not be undervalued in the genesis of endocarditis.

Pathologic Anatomy.—Anatomically, verrucose, sclerotic and ulcerative endocarditis are to be differentiated. The first two forms of endocarditis run a benign course in childhood as a rule, though they only rarely lead to complete recovery, while the last form almost always leads to a fatal termination.

The anatomical picture of endocarditis in childhood hardly differs from that of adults. In the verrucose form rose-red outgrowths, not unlike verruca acuminata, arise as a result of vegetation of the fibrous layer of the endocardium, on the side of the valves facing the ostium,

with destruction of the covering epithelium. These may be totally reabsorbed or undergo a fibrous change, with contraction of the valvular apparatus, from which stenoses of the ostia and inability of the valves to close may result (chronic endocarditis; valvular heart lesion).

The ulcerative form is different, in that the granulation tissue, rapidly forming upon the valves, is broken up by ulceration, which may lead to destruction of the valves, erosion of the chordæ tendineæ and papillary muscles, and finally to purulent myocarditis. If infectious matter from the endocardium is carried off by the blood, abscesses may be caused by emboli in the most remote organs, the kidneys, spleen, liver, lungs or central nervous system.

Embolic processes may also occur in verrucose endocarditis, from separation of particles of the valvular excrescences, which produce hæmorrhagic infarcts. Finally, that papillary vegetation and ulcerative processes may also occur on the parts of the endocardium apart from the valves (*parietal endocarditis*) should be noted.

Bacteriologically, it should be mentioned that a large number of different microbes have been found in verrucose and ulcerative endocarditis, staphylococci, streptococci, typhoid bacilli, pneumococci, bacillus pyogenes fœtidus, gonococci, etc. The occurrence of endocarditis depends, according to Fränkel and Sänger, upon the blood in the left side of the heart containing more oxygen, which better supplies the oxygen needed by the above-mentioned micro-organisms than does the venous blood of the right side of the heart.

Normally small nodules are frequently found on the valves of the heart, in newborn and young children, during the first year of life, which are usually situated near the free edges of the valves and must be noted here only because they have for some time been mistaken for endocarditis excrescences and because Bouchut, Labadie and Lagrave, who found them in children dead from diptheria, considered them as diphtheritic endocarditis. These conditions, wholly without clinical significance, are to be differentiated anatomically from the round nodules of Albini, as big as the head of a pin, seated at the free edge of the venous valves, 20 to 30 in number, and from the peculiar valvular hæmatomata which are, according to Haushalter and Thiry, small spherical tumors, dark violet in color, varying from the head of a pin to a grain of rice in size, located some distance back from the free edge of the valve, from 3 to 10 in number.

(*b*) SPECIAL PATHOLOGY OF ENDOCARDITIS

Clinically acute and chronic endocarditis of children are to be differentiated, the acute form being subdivided into a benign and malignant endocarditis. But acute malignant endocarditis is a great rarity in childhood.

1. *Acute Endocarditis in Children*

Symptoms.—The most important and in many cases the only clinical symptom is a systolic murmur at the apex, corresponding to the mitral valve, with but slight transmission to the rest of the precordial region at first. As valvular endocarditis in childhood affects the mitral valve almost exclusively, the localization of the murmur is easily understood. The especial anatomical relation of endocarditis in children to the aortic tip of the mitral valve depends upon the exceptionally great tension of this part of the valve, when the ventricles contract with systole. Murmurs localized at the aortic ostium are very rare in childhood. Among 33 cases of endocarditis observed by Hochsinger the aortic valves were affected only in four.

Goodardt found among 256 cardiac affections of childhood

Affections of the mitral valves in 142,
Affections of the aortic valves in 11,
Affections of the aortic and mitral valves in 22,
and a doubtful diagnosis in 56.

The production of the murmurs in acute endocarditis in childhood may be due to roughness, papillary excrescences, or diminished tension of the valves, chordæ tendineæ and papillary muscles, from inflammatory changes. The possibility of a relative inability of the mitral valves to close in acute parietal endocarditis is not to be denied, although the extraordinarily resistent heart in childhood possesses no especial tendency toward the rapid development of dilatation. The production of the murmur in valvular endocarditis in children is preceded in most cases by a weakening, impurity or dull quality of the first sound of the heart; Potain even considers this sound anomaly the essential auscultatory phenomena of mitral endocarditis of childhood; and explains the murmurs which may appear later as extracardial cardiopulmonary murmurs (see p. 446), a point of view which is incompatible with the great constancy of this murmur, with its continuation during respiratory pauses and during expiration, and with its accentuation during crying, as has been observed by Hochsinger repeatedly.

Although, acoustically, as a rule, the murmurs of endocarditis in childhood represent a low blowing or whistling sound joined to the first heart sound, musical murmurs which may completely conceal the first sound of the heart have been observed, even when the endocarditis has lasted only a short time. Sometimes a mitral murmur which has appeared rapidly is heard more loudly in children in the third intercostal space on the left side than at the apex, if as the result of sudden inability of the mitral valve to close, the murmur becomes accentuated because of the backward movement of blood toward the left auricle. The mur-

mur of acute mitral endocarditis of children is only transmitted imperceptibly or not at all to the aortic and tricuspid ostia.

Acute endocarditis of the aortic valves in children also gives rise to a systolic murmur, with its point of maximum intensity at the second intercostal space to the right of the sternum, and is the result of unevenness of the valvular ring and the edges of the valves, due to verrucose efflorescences upon the valves. Only when true insufficiency of the valvular apparatus occurs, *i.e.*, after the process has lasted some time, are diastolic murmurs heard at the aortic ostium. On the whole, diastolic aortic murmurs are very rarely found during the first years of life, with acquired heart affections.

Percussion of the heart gives fewer positive signs than auscultation in acute endocarditis in children, because an increase in the heart dulness may not be certainly shown clinically for a long time, or may even be absent during the entire course of the disease, in cases which end in recovery. This is in part due to the extraordinary resistance of the cardiac muscle in children, which prevents dilatation of the heart cavities; in part it depends upon the difficulty of estimating, by percussion, slight grades of dilatation of the left auricle which next comes into question in acute mitral endocarditis of childhood. An increase in the size of the right side of the heart which appears rapidly in endocarditis of adults is absent for a long time in the endocarditis of children, because the compensatory overwork of the left ventricle causes it to empty itself so thoroughly into the aorta that congestion in the pulmonary circulation is prevented for a long time. The first and only sign of a backward action upon the right side of the heart in the acute mitral endocarditis of children is as a rule accentuation of the second sound at the pulmonary area only, and this symptom may be lacking for a long time. Thus, in children, there may be a stage lasting weeks in which a systolic heart murmur is the only physical sign of acute endocarditis.

The functional symptoms of the acute endocarditis of childhood are very varied. Subjective symptoms are absolutely absent in some children. Sometimes older children complain of pain in the precordial region or pain piercing the left side of the chest suggesting pleurisy, or the pains blend with rheumatic pains which often exist at the same time. Oppression in the chest, anxiety and restlessness are usually present only when pericarditis accompanies the endocarditis. One symptom frequently present is a sort of nervous shortness of breath, which appears with bodily exertion, talking, crying or the slightest psychical excitement, and cannot be explained by the other objective findings.

Acute endocarditis in children always begins with a rise of body temperature. If it complicates another febrile disease, it produces an

exacerbation of the fever, or if the temperature of the fundamental disease has already reached normal, it rises again with the endocarditis. No certain type of fever prevails. The temperature may return to normal rapidly and shoot up again with new attacks, frequently marked by the recurrence of rheumatic pains. Then there are cases of idiopathic endocarditis in early childhood in which the rise of temperature is slight, sometimes only ephemeral, and a cardiac murmur, only appearing later, shows that the fever observed previously was the initial fever of endocarditis.

The pulse-rate is always accelerated but its rhythm usually remains unchanged.

Diagnosis.—If a cardiac murmur appears in the course of or as a result of polyarthritis or the infectious diseases, with a rise in body temperature, remains permanently and is not modified by pressure with the stethoscope and by respiratory pauses, it always points to acute endocarditis. Difficulties may arise in the interpretation of the murmurs, as it may sometimes be necessary to distinguish the murmur of simple fever from organic cardiac murmurs (see p. 446). Sometimes the differentiation between acute endocarditis and acute dilatation of the heart, with relative insufficiency of the mitral valve, may be difficult in childhood. With regard for the high compensatory tendency of the cardiac muscle in children, mentioned in the symptomatology of endocarditis in childhood, as opposed to the acute changes of endocarditis, when cardiac dilatation, together with a systolic murmur, appears rapidly, the decision will always result in favor of a dilatation insufficiency of the mitral valve. The same thing will also come into question in nephritis, chronic pneumonia, cardiac insufficiency as the result of septic diseases, and chronic myocarditis with acute cardiac insufficiency, in children. Several cases of heart murmurs, reported in literature in little children without anatomic changes of the valves, which on that account were considered accidental, depended upon relative insufficiency of the mitral valve.

The murmur of endocarditis soon after its appearance is only rarely so harsh in childhood that it can be mistaken for the friction murmur of pericarditis. On the contrary, however, pericardial friction murmurs may be so soft in children that it is difficult to tell them from endocardial murmurs. As, besides, in a considerable number of children endocarditis appears simultaneously with pericarditis, the murmurs heard in auscultation may sometimes be very difficult to explain, in the very acute cases of inflammatory cardiac affections of children. The superficial and noncontinuous character of the pericardial friction murmurs which is not completely concealed by the phases of the heart, their slight transmissibility and their accentuation when pressure is made with the stethoscope are also observed. Sometimes the exist-

ence of a pericarditis in addition becomes plain from the fact that the murmur, in parts of the thorax far from the precordial region, is quite different in character from that heard at the apex and over the left ventricle. That the pericardial friction murmurs are frequently only heard in children during ventricular systole has already been mentioned in the section on pericarditis. The diagnosis of acute endopericarditis of childhood becomes more probable when, in spite of the essential symptoms of endocarditis and in spite of a strong pulse, with loudly audible murmurs, the apex-beat appears to be weakened.

Course and Prognosis.—Acute endocarditis of childhood may lead to complete recovery or to chronic changes in the valvular apparatus of the heart, or in rare cases may end in death from complications. Malignant or ulcerative endocarditis, which will be briefly described later, always leads to a fatal termination rapidly.

According to the observations of Steffen, Hochsinger and Weill, an acute endocarditis in children, rheumatic or scarlatinal, or parietal endocarditis also, may completely disappear within a few weeks, with the gradual disappearance of the murmurs. Affections of the aortic valves do not show this involution ability. Most inflammations of the valvular endocardium lead to permanent cardiac lesions, from contraction of the vegetations, in part, too, from calcification of the valvular excrescences and of the valves themselves.

Death may result in the benign form of endocarditis also, from association with pericarditis, pericardial adhesion or embolic processes in the brain. One of the important resulting conditions in childhood is cerebral embolism, producing hemiplegia. In a case of Hochsinger's, affecting a child of three and a half years, right hemiplegia occurred toward the end of the second week of an acute scarlatinal endocarditis, marked by a low blowing murmur.

The **prognosis** as to life, may in general be favorable if there are no points to support the presence of pericarditis at the same time. While most cases of endocarditis of childhood lead to permanent valvular lesions, nevertheless, death hardly ever occurs in uncomplicated cases, during childhood, but only in later life.

Malignant (Ulcerative) Endocarditis

As a result of the high virulence of the caustive micro-organisms severe pyæmic or typhoid symptoms occur on the one hand; while, on the other, the rapid destruction of portions of the inflamed valves and endocardium brings about a violent eruption of serious symptoms of cardiac insufficiency, among which acute dilatation, with loud murmurs at all of the ostia, comes first. S. Adams was able to collect 47 cases of septic endocarditis in childhood, with recovery in three cases.

When heart murmurs are also present, the diagnosis can sometimes

be made in childhood, as was done in two cases by Hochsinger. Murmurs are absent very frequently (Jacobi) and are replaced by embryocardial or gallop-rhythm which may cause insurmountable obstacles to the diagnosis. This is the rule in early childhood especially, when the seat of the ulcerations is parietal and the endocarditis is associated with pulmonary affections.

From an often puzzling illness, resembling typhoid, irregular malaria, miliary tuberculosis or pyæmia, children with ulcerative endocarditis may suddenly develop a serious cardiac symptom-complex, with the appearance of loud cardiac murmurs. This is the case if parietal ulcerations advance into the myocardium and produce perforation of the septum or cardiac and valvular aneurysms. Under these conditions the symptoms of septic emboli appear rapidly. Capillary emboli of the skin, with purulent or hæmorrhagic efflorescences, paralyses appearing suddenly, intestinal and renal hæmorrhages, widespread areas of pulmonary dulness, with high fever and loss of consciousness, mark the embolic, septic character of this morbid process which always leads to death.

According to Baumgarten, in the majority of cases, the staphylococcus aureus and the streptococcus pyogenes act together to produce ulcerative endocarditis, the chief part as regards the ulcerations being attributed to the latter, however. But the colon bacillus, different proteus forms and other micro-organisms have also been found.

Malignant endocarditis may attack children in perfect health or it may also develop in the course of other infectious diseases, caused by the microbes of the disases (typhoid fever, streptococcus pneumonia, ulcerative colitis, erysipelas). Ulcerative endocarditis may also be joined to chronic endocarditis and pericarditis.

The **duration** of malignant endocarditis varies. There are forms in childhood, violent at the beginning, with high fever, leading to death within a few days with septic symptoms. Other cases drag along for weeks with constantly repeated attacks of embolism and fever, until death occurs with symptoms of exhaustion. Remarkable and characteristic of the great tenacity of the cardiac musculature of children is the rarity of œdema and decided venous congestion, even in these serious forms of acute heart disease.

No effective treatment of malignant endocarditis exists. Hydrotherapy and stimulants should always be tried. In any case the treatment of insufficiency of the cardiac muscle should be begun very early (see p. 517).

Treatment of Acute Benign Endocarditis.—As in pericarditis, a prophylactic treatment of endocarditis in childhood may be mentioned; salicylic preparations should be given for the slightest rheumatic symptoms of children and complaints of pain in the joints and

muscles in later childhood should never be allowed to pass without attention.

The drug treatment of the disease, after it has developed, is to be restricted whenever possible. Absolute rest and the permanent application of cold, the prevention of bodily and psychic excitement, nonstimulating diet, an antiphlogistic general regime in cases with fever (without internal antipyretics) with especial attention to hydrotherapy [cold sponge baths at 25° C. (87° F.)] suffice for simple cases. When rheumatic attacks also occur, the use of salicylates is indicated, but only long enough to overcome the joint pains and swelling. So, too, digitalis should be administered as sparingly as possible in the acute endocarditis of childhood. Blisters and cauterization, as recommended by French writers, are superfluous and often injurious procedures. When the heart action is excited, bromides are given, and when the pulse is of high tension, sodium or potassium iodide, in doses of one to three decigrams a day. With decided pains in the chest and attacks of dyspnœa in older children, codeine, dionine [1 to 2 cg. ($\frac{1}{6}$–$\frac{1}{3}$ gr.) daily] or morphine [$\frac{1}{2}$ to 1 cg. ($\frac{1}{12}$–$\frac{1}{6}$ gr.) daily] must be administered. Recently the treatment of acute heart affections of children by opium has been warmly advocated by Morison.

2. *Chronic Endocarditis and Acquired Heart Lesions of Childhood.*

The most frequent cause of heart lesions in children is acute endocarditis. Among 478 children between 2 and 15 years of age observed during fifteen months, Weill found 25 (5 per cent.) with valvular heart lesions. This condition occurs very rarely before the fifth year but frequently in later childhood, though only half as frequently as in adult age.

Rheumatism plays the principal part in the **etiology**, leading in children, sometimes without previous symptoms of endocarditis, to chronic changes upon the ostia and valves of the heart. In childhood, doubtless, too, there occurs a chronic primary sclerosis of the valvular endocardium which leads to stenoses of the ostia, believed by Potain and Teissier to be associated with tuberculosis. A certain family predisposition to chronic mitral endocarditis cannot be shown. Chronic arteriosclerosis, lead poisoning and diabetes mellitus do not play any part in the occurrence of cardiac lesions in childhood. Only syphilitic affections of the endocardium sometimes lead to heart lesions in children. In the majority of cases chronic endocarditis is the result of an acute endocarditis which has recovered by cicatrization.

Acquired valvular lesions of the aortic ostium develop almost exclusively in later childhood, though aortic insufficiency, without other cardiac anomaly, with the ductus Botalli closed, showing that the lesion is acquired, has been seen by Seiffert in a child of eighteen months.

Hauser observed in a child of fourteen months true stenocardia cordis, due to cicatrizing endocarditis at the insertion of the aorta, below the ostium, with totally uninjured valves and consecutive, very considerable hypertrophy of the heart.

Pathologic Anatomy.—No essential differences exist between chronic endocarditis in children and in later life, except for the especial predilection of the mitral valve in childhood. According to the investigations of Steffen and Hochsinger, one affection of the aortic valve occurs to 18 mitral lesions in childhood. Great compensatory hypertrophy of the left side of the heart appears much more rapidly in childhood, especially with mitral insufficiency; while dilatation is only observed after some time and then is always accompanied by considerable hypertrophy. Hypertrophy and dilatation of the right side of the heart occur only in severe cardiac lesions of children, which have lasted a long time and are accompanied by considerable functional disturbances, excepting lesions of the pulmonary valves, which depend upon congenital anomalies chiefly.

Symptoms.—The physical signs depend upon the seat of the lesion, its effect on the circulation and cardiac muscle and the manner in which the myocardium reacts, and are on the whole concealed by symptoms well known from the pathology of later life.

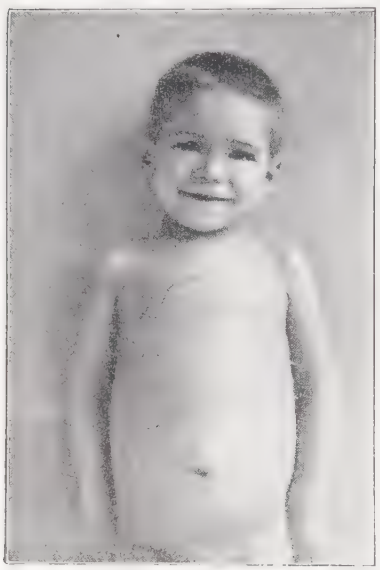
Some peculiarities in the auscultation of children should be mentioned. The murmurs of valvular heart lesions in children are usually very loud. In childhood, in contrast to later periods of life, it is very exceptional for a serious lesion to run its course without apparent murmurs. Besides, cardiac murmurs in children have as a rule two points of maximum intensity, one upon the anterior chest wall corresponding to the seat of the valvular lesion and the other in the back, to the left or right of the vertebral column (see also p. 445). In small children with decided cardiac hypertrophy the murmur is not rarely transmitted to the sacrum or to the head and extremities.

When cardiac lesions are combined in childhood, an excessive increase in the size of the heart very frequently develops, with increase in its cavities (*cor bovinum*). In such cases a marked forward arching of the left side of the heart occurs, with an intense vibration in all the intercostal spaces, a forceful, very diffuse apex-beat and not rarely a very enormous area of heart dulness. As a rule, then, the heart occupies a more horizontal position, with the right ventricle, in its large circumference, lying directly against the diaphragm. An epigastric or abdominal pulsation is visible from a distance. In such conditions the heart murmurs are also heard in the epigastrium and over the entire abdomen. Radioscopic examination best shows the relations of the heart and its separate parts in position and size.

Several *physical signs* which are frequent and of special importance

in the heart lesions of adults are rare in childhood and of less significance. Among these is arrhythmia, which may constantly be absent absolutely in serious fatal cardiac lesions of childhood; which is, however, more frequently found in children whose hearts are unaffected. Accentuation and a metallic quality of the second sound at the aortic area is very rare in cardiac lesions of children; on the other hand an accentuation of the second sound at the pulmonary area is more frequent with hypertrophy of the right side of the heart. Reduplication of the second sound at the base of the heart, more frequently found in mitral stenosis of adults, has no significance in early childhood. True venous pulse, dropsy and decided hypertrophy of the visceral organs from congestion are rare in early childhood, as a result of the extraordinarily strong compensatory apparatus of the cardiac musculature (see p. 442).

Fig. 109.



Boy of five years with uncompensated mitral insufficiency, considerable eccentric hypertrophy of both sides of the heart (cor bovinum) and decided tumor of the liver, due to congestion.—The outlines of the heart and liver dulness are marked.

As regards the *functional symptoms* of acquired heart lesions in children, stress should be laid upon the extraordinary subjective tolerance of children. Most cardiac lesions in children persist until puberty with good compensation, and especially the years before the child goes to school are very free from subjective disturbances on the part of the diseased heart. Frequently loss of compensation, with all its well-known functional injuries, first appears at the age of puberty, simultaneously with more intense mental demands upon the children. Here the favorable relation between the width of the blood vessels and the heart, already mentioned on page 442, has become shifted, to the detriment of the heart.

According to Perret's statistics, 21 per cent. of acquired heart lesions of children were free from subjective symptoms; 21 per cent. showed slight functional disturbances and 42 per cent. had moderate or severe symptoms of loss of compensation. Almost one half of the cases in childhood run their course without subjective symptoms.

Cardiac lesions are, on the whole, better borne during childhood than in later life. The most essential subjective disturbances are dyspnœa, palpitation and a tendency to bronchial catarrh. Apart from the rare syphilitic affection of the heart, stenocardial attacks are not observed in childhood because of the absence of arteriosclerosis.

Often children with acquired heart lesions show a peculiar cachexia, which Germain Sée has correctly described as the chlorotic form of juvenile cardiac lesion. The children are strikingly pale and wan, like young girls with chlorosis, suffer from headache and dyspepsia; are short of breath, indolent, very capricious and irritable and show a slight cyanotic discoloration of the mucous membrane. According to Hochsinger's experience children with cor bovinum are of this type very constantly. Striking changes in the relations between white and red blood corpuscles are lacking in chronic acquired heart lesions of childhood, in contrast to the congenital lesions.

Diagnosis.—The diagnosis is divided into two parts, the general diagnosis of a cardiac lesion and the recognition of the seat of the lesion. As functional disturbances are lacking in half of the cases, the consideration of the physical signs in children is of double importance. (For the general differential diagnosis between congenital and acquired heart lesions, see page 449.) The diagnosis of the addition of chronic endocarditis to congenital disturbances in the development of the heart, a frequent occurrence, can be made sometimes from the changes or modifications of the physical signs in conjunction with an increase of the subjective symptoms.

Mitral Insufficiency.—This, the most frequent cardiac lesion of childhood, is characterized by the development of hypertrophy of the left side of the heart, especially of the left auricle, in conjunction with a systolic murmur with its point of maximum intensity at the apex. The murmur is transmitted to the left axilla and to the back. Pure forms of this cardiac lesion are often seen for a long time in childhood.

Mitral Stenosis.—This frequently accompanies mitral insufficiency in later childhood, and is characterized by a diastolic, sometimes split murmur at the apex. In childhood especially the transition of a mitral insufficiency into a pure mitral stenosis has been observed, with the disappearance of the systolic and the substitution of a diastolic murmur at the apex. It is, however, a well known fact, corroborated by observations during childhood especially, that diastolic murmurs may be absolutely absent in mitral stenosis (Jacobi).

Aortic Stenosis.—In these acquired heart lesions, in childhood an inflammatory adhesion of two or all of the semilunar valves is usually found. This affection is found only in later childhood and very rarely alone, being almost always associated with inability of the aortic valves to close. The physical signs are a rough systolic murmur over the aortic area and hypertrophy of the left ventricle.

Aortic Insufficiency.—This is always combined with stenosis of the aortic ostium and is the gravest form of heart lesion in childhood. Children with aortic insufficiency rarely live beyond puberty. Sudden death not rarely occurs. The aortic tip of the mitral valve is almost

always affected also by the chronic inflammation so that, when this has lasted some time, a combined lesion results, with an unfavorable prognosis. The pathognomonic sign of aortic insufficiency is a diastolic murmur with its point of maximum intensity in the second intercostal space to the right of the sternum, which is transmitted downward to the xiphoid process, but is also heard, though weaker, toward the neck.

The valvular lesions and ostium stenoses of the right side of the heart have been described with the congenital heart lesions, since they originate for the most part during fœtal life. It should be noted, however, that tricuspid insufficiency, which produces a systolic, usually whistling murmur over the xiphoid cartilage, is sometimes joined to mitral insufficiency in the stage of loss of compensation, as a relative insufficiency.

Course.—A peculiarity of juvenile heart lesions, especially of mitral insufficiency, consists in their long latency, their extraordinary compensatory ability and their slight mortality during childhood. In aortic insufficiency, the situation is less favorable. Recovery from well-developed cardiac lesions has been repeatedly noted in children, even after the lesion has lasted for years (Roger, Trousseau, de Gassicourt, Sanné, Andrew, Gerhardt, Ovazza, Hochsinger). In the majority of cases severe disturbances of compensation, with œdema, diminished secretion of urine, pulmonary catarrh from congestion and the production of infarcts, only develop after puberty. But all these symptoms occur in childhood also in complicated lesions, with frequent recurrence of endocarditis, although they are rare before the end of the fourth year.

Marfan considered mitral endocarditis of childhood so benign that he always blamed the appearance of insufficiency of the cardiac muscle, with the clinically established symptom-complex of mitral insufficiency, upon some accompanying affection. In this relation latent pericardial adhesion is first to be noted, and secondly the presence of congenital heart lesions also.

Treatment.—The treatment of acquired valvular heart lesions of childhood differs little from that employed in later life, as the children suffering from acquired lesions almost always belong to later childhood. A certain hygienic care of those children who show no real functional disturbances is of importance. All overexertion of the heart from bodily or mental demands is to be avoided. Athletic sports and swimming exercises are forbidden, but on the contrary regular movements of the muscles, by walking in the open air, are advised. Incorrect carriage of the body should be corrected. Nourishment should be abundant, without too much liquid and with no acrid substances. Tea, coffee, alcohol and tobacco must be absolutely forbidden.

As all the infectious diseases may lead to new attacks of endocarditis a prophylaxis, doubly careful in this respect, is to be employed for children with heart disease.

The choice of occupation for children with heart disease is important. For children of the middle class business comes first; for those of the working class occupations are to be chosen which are not attended by continued severe bodily exertion and do not cause leading a constantly sedentary life. They should become barbers, glaziers or upholsterers.

House and clothing need attention to prevent the recurrence of rheumatism. Dry airy apartments, in the sunshine at least part of the day, are of importance for children with heart disease; their clothing should be warm during winter, but heavy goods should be avoided. In summer all those materials which imbibe moisture should be avoided (silk and linen). Cassel advises woolen clothes for children with heart disease for at least several years after the last attack of rheumatism.

Balneotherapy in the form of slight hydrotherapeutic measures, partial sponges at from 20° to 16° C. (68° to 60° F.); half baths from 26° C. (78° F.) down; cold water tubes on the heart region twice or three times daily for an hour, and carbonic acid baths, either natural at Nauheim or Franzensbad, or artificial at home, assists the prophylactic and dietetic treatments.

For cardiac weakness, with loss or compensation, reference should be made to the principles explained later, on page 517.

6. MYOCARDITIS

Pathologic Anatomy.—In childhood as in all periods of life, parenchymatous and interstitial myocarditis are distinguished anatomically. Parenchymatous processes often appear acutely in the course of severe infectious diseases (diphtheria, typhoid fever, scarlet fever, whooping-cough) in which, as the final stage anatomically, fatty degeneration (yellow atrophy) occurs. In the acute parenchymatous processes the muscular tissue of the transverse bands appears to be lost, the muscle fibres are filled with a finely granular mass, show hyaline degeneration or are broken into pieces and in many places replaced by fatty droplets. Pure parenchymatous myocarditis, without affection of the interstitial tissue, has been noted in childhood by Bouchut, Barjon, Weill and Janot. In the majority of cases, however, primary parenchymatous myocarditis is associated with interstitial changes also.

The heart in parenchymatous myocarditis is soft, flaccid, pale, pliable and crossed by yellow stripes or hæmorrhages in the region of the apex.

Parenchymatous affections of the heart muscle in childhood very frequently accompany all possible infections and are equally as frequent in infancy as in later childhood. Sudden death during many of the infectious diseases of children depends upon parenchymatous myocarditis.

Much more rare in childhood are interstitial myocarditis processes which may take an acute or chronic course. Acute interstitial inflammations of the heart muscle arise in children either by continuation of inflammatory processes to the pericardium or endocardium, or embolically, by the introduction of microörganisms into the blood vessels of the myocardium. The most severe form of interstitial myocarditis is purulent myocarditis which occurs in the septic forms of scarlet fever, measles and diphtheria, and in osteomyelitis of children also, which may lead to abscess formation in the wall of the heart and in the septum, to perforation into the cardiac cavity and pericardium and to the development of acute aneurysms of the heart.

In interstitial myocarditis the cellular tissue between the muscle fibres shows cellular, *i.e.*, purulent, infiltration either circumscribed to certain areas or diffuse. In the chronic forms callosities are frequently found which may lead to chronic aneurysms of the heart and sudden rupture. Here also belong the syphilitic callosities, gummata in the later stage of hereditary syphilis, and scattered cases of tuberculous myocarditis.

The mixed form of myocarditis is most frequent, affecting all the tissues composing the wall of the heart. This may be circumscribed or diffuse. In the latter the muscle fibres, connective tissue, cardiac nerves and blood vessels of the cardiac wall show inflammatory changes. The worst cases clinically are the result. In diphtheria especially a perineuritis of the cardiac nerves has been found very frequently. Often the small vessels have been closed by vegetation of the intima or by thrombosis, causing hæmorrhages in the vicinity.

The nodular form of interstitial myocarditis is usually purulent, the pathology of which has just been described.

Eppinger has termed toxic myolysis of the heart in diphtheria an œdematous infiltration of the myocardium, breaking the course of the muscle fibres, with vacuolization and complete dissolution. He believes that the diagnosis of cardiac death from diphtheria can be made with certainty from these changes in the heart.

As regards the pathogenesis of the various forms of myocarditis, different microörganisms which produce inflammation have been found in the wall of the heart itself, in a series of cases of the interstitial forms (pus cocci, typhoid bacilli, bacillus pyocyaneus). Doubtless, however, the toxins of the infectious diseases play the principal part, in diphtheria, scarlet fever and typhoid fever especially, the last only rarely exerting in childhood that serious influence upon the heart which it has upon adults.

Winogradow found important pathologic anatomic changes in the automatic ganglia of the heart in 22 cases of congenital syphilis of infants. With interstitial growth of the connective tissue and changes

in the blood vessels, severe degenerations of the ganglion cells have frequently been found. B. Fischer described serious syphilitic changes in the myocardium with aneurysmic dilatation of the conus venosus, in a boy of five years.

Symptoms and Course.—The symptoms of acute parenchymatous myocarditis consist of diminution in the power of the heart, which is recognized by marked weakness of the pulse, impossibility of feeling the apex-beat and low heart sounds with embryocardial ryhthm. The pulse is as a rule enormously accelerated and the second sound of the heart is frequently inaudible. These children show high-grade dyspnœa, a deep pallor, cold cyanotic extremities and peripheral parts of the body, in contrast to the rest of the body surface, which usually feels very hot, because the disease produces fever. These children are tormented by a peculiar vexing restlessness which cannot be bettered and show rapid respiration, especially high in the chest, combined with movements of the alæ nasi with respiration.

In rare cases of chronic parenchymatous myocarditis after infectious diseases, belonging to later childhood, there is retardation of the pulse, with arrhythmia and irregular respiration, at times abnormally slow, and then again very rapid. The rapid rise in the pulse-rate and respiration, with the slightest bodily or mental excitement is characteristic of this form of myocarditis in children.

The symptomatology of parenchymatous myocarditis is on the whole not very well outlined, so that the disease in many cases remains unrecognized during life (Zuppinger).

Still more uncertain is the symptom-complex of acute interstitial myocarditis. Symptoms of cardiac weakness and dilatation are combined with the serious symptoms of the fundamental disease. When, in the course of an acute infectious disease, severe symptoms of dyspnœa, weak rapid pulse and cyanosis develop gradually, unexplained by any intercurrent pulmonary disease, the diagnosis of the presence of myocarditis is justified, though the question whether it be an interstitial or parenchymatous myocarditis remains undecided.

As in all morbid processes which lead to relaxation of the heart muscle, dilatation of the cardiac cavities (sometimes high-grade, Cruchet) with relative inability of the valvular apparatus to close, thus producing murmurs, may also occur in myocarditis. The diagnosis of the myocarditic processes which follow acute and chronic endopericarditis, symphysis pericardii and congenital heart lesions cannot be made. Loss of compensation and death from chronic affections of the heart in childhood are often the result of myocarditic processes which have appeared during new attacks of rheumatism or in the course of intercurrent infectious diseases.

Intracardial thrombosis, with emboli, has repeatedly been observed

in the various forms of myocarditis, especially diphtheritic myocarditis (Degny and Weill, Leyden).

The symptomatology of *diphtheritic myocarditis* (cardiac death from diphtheria) deserves a brief special description. After the local symptoms of diphtheria have disappeared, rapid pulse, pallor and dyspnœa appear, with bodily and mental excitement, a condition of nervous vomiting and a tendency to fainting even with slight movements of the body. Examination of the heart shows diffuse precordial undulation (trembling of the heart), frequently dilatation of the heart with systolic murmurs. Death occurs either slowly, with gradual diminution in strength, or suddenly.

Prognosis.—Diphtheritic myocarditis is fatal in more than half of the cases. The appearance of conditions of collapse and fainting during convalescence from the infectious diseases is always unfavorable and depends upon diffuse myocarditis. Myocarditis in childhood is always a dangerous affection, whether because it predisposes to sudden death, or because it causes contraction of the interstitial connective tissue of the wall of the heart, the effect of which is only noticeable in later life.

Diagnosis.—Functional heart symptoms are differentiated with difficulty from myocarditic symptoms, in the febrile infectious diseases. The persistence of cardiac symptoms for a long time, sometimes longer than the period of fever, points to myocarditis. The symptom-complex of diphtheritic myocarditis is sometimes hardly to be distinguished from that of a diphtheritic vagus paralysis.

Myocarditic heart collapse occurs in typhoid fever, but frequently the diagnosis is made incorrectly, when the collapse depends upon intestinal hæmorrhage or perforation.

Myocarditis due to scarlet fever is rare and is not to be mistaken for dilatation of the heart from nephritis, which has already repeatedly been mentioned as occurring in scarlet fever.

Treatment.—In all the infectious diseases, great stress should be laid from the very beginning upon the condition of the heart; modern antipyretic drugs and the administration of alcohol are to be absolutely avoided, while on the other hand hydrotherapy and measures for as abundant nourishment as possible are to be used. When symptoms of myocarditis appear, every superfluous movement and every psychic excitement should be prevented, while the ice bag or other cooling apparatus should be constantly applied to the cardiac region. Camphor should be given for attacks of fainting and collapse. Calamet advises the subcutaneous employment of caffeine sodiobenzoate or sodiosalicylate [0.25–1.0 Gm. (4 to 15 gr.) daily] or sparteine sulphate [0.4 Gm. (6 gr.) to 10 c.c. ($2\frac{3}{4}$ dr.) aq. destill., given hypodermatically once or twice a day] for the myocarditis of typhoid fever. Treatment with digitalis continued for some time should be begun when symptoms

of chronic myocarditis appear. After myocarditis has run its course, it is urgently advised to carefully watch the children, to prevent excessive bodily and mental exertion.

FATTY DEGENERATION OF THE HEART

This occurs in childhood as an acute and a chronic disease. As partial symptom of an acute fatty degeneration of several parenchymatous organs and of the transverse musculature, acute fatty degeneration of the heart is found in the so-called Buhl's Disease of the Newborn, in Winckel's Disease and in malæna neonatorum. It is also found in childhood in phosphorus poisoning, very wide spread burns, purpura fulminans and after severe hæmorrhage. The chronic form of fatty degeneration of the heart is found in pernicious anæmia, leukæmia, high-grade infantile atrophy and abscesses of long duration. Many of the heart affections in older children, especially chronic pericarditis and pericardial adhesion, lead to fatty degeneration of the cardiac musculature. The symptomatology is covered by that of chronic myocarditis.

7. INSUFFICIENCY OF THE HEART MUSCLE (MYASTHENIA CORDIS)

Those functional disturbances of the child's heart should be included here which depend upon permanent organic injury to the power of the heart, whether there is question of primary organic injury to the heart muscle or of myopathic results of mechanical obstruction to heart action (cardiac lesion, affection of the pericardium). Doubtless, as a result of the favorable relations of the heart muscle of children, the functional disturbances belonging here are rarer than in later life. Affections of the kidneys and lungs, the chief causes of myasthenia cordis in adults, come into question in childhood relatively little, precisely on this account. Whooping-cough alone, exceptionally, when it runs an especially severe course, leads to acute insufficiency of the heart, combined with dilatation (Silbermann, Hauser, d'Espine and Picot).

The chronic valvular lesions of the heart possess a high compensatory tendency in childhood while, on the contrary, pericarditis and pericardial adhesion in children form the chief causes of this condition (Cadet de Gassicourt, Marfan, Weill). The pathogenesis of cardiac insufficiency in children is therefore dependent essentially upon toxic myocarditis and mechanical obstruction to heart action by cardiac lesions and symphysis pericardii, which are associated with sclerosis of the myocardium and parenchymatous myocarditis in many cases. The growth of fat about the heart, coronary sclerosis, the effects of alcohol and tobacco do not enter into the question.

Symptoms.—The symptoms of insufficiency of the heart muscle (asystolia of the French) are not so outspoken in childhood as in older individuals. Considerable dilatation of the right side of the heart and

functional tricuspid insufficiency are only noted in a small number of children with heart disease. Anasarca also is commonly absent or as a rule first appears late in the disease, some weeks or days before death. Pulmonary catarrh from congestion and diminished secretion of urine occur more frequently and earlier than anasarca, but are less prominent on account of the decided symptoms of congestion in the liver which developed much earlier. In childhood the congestion of heart disease affects the liver much more than the other organs, although it is difficult to understand why (Fig. 109). The liver is the single organ of the child in which symptoms of cardiac congestion are plainly observed, in the majority of cases.

While myasthenia cordis does not affect the child's pulse in any characteristic manner, as it does in adult's (arrhythmia, gallop-rhythm), the symptoms in the respiratory organs are more severe. Often a disproportion exists between the violent dyspnœa and the very slight changes in the heart as shown by physical signs. The external habitus of children suffering from advanced myasthenia cordis, after it has lasted some time, hardly differs from that of the adult, except that dropsy is usually absent in children. Cyanosis of the peripheral parts of the body, venous stasis when the condition has lasted a long time and clubbed fingers are prominent symptoms.

Briefly then, in the majority of cases, there are symptoms of moderate venous stasis, with marked disturbance of the hepatic circulation and only exceptionally symptoms of tricuspid insufficiency, with anasarca, but always decided dyspnœa (over 40 respirations to the minute).

Prognosis and Course.—While insufficiency of the heart muscle, under suitable treatment, may be well borne for years in adults, since it is possible within certain limits again and again to restore the insufficient cardiac muscle for a while, myasthenia cordis in childhood leads to death relatively rapidly, even if the cardiac insufficiency occurring during the infectious diseases is considered completely apart from the acute conditions. The opinion as expressed by Weill that compensated heart affections in children are more benign than those of adults, although those with loss of compensation are much more serious in the former than in the latter, is correct.

The subjective symptoms of insufficiency of the heart muscle are less severe in children than in adults. Painful palpitation, feelings of oppression, piercing pains in the cardiac region are rare symptoms even in advanced cases, while older children, on the contrary, complain of debility and gastro-intestinal disturbances, stomach ache, regurgitation, loss of appetite, constipation or diarrhœa. With chronic cardiac diseases of children symptoms of myasthenia usually appear absolutely outspoken, in contrast to myasthenia of adults in which initial stages, mild often for years, are observed.

Hochsinger considers that insufficiency of the heart muscle in obese older children has received little attention. It is not as if the accumulation of fat upon the heart itself would be an obstacle to its contraction, but rather the disproportion between volume and strength of the heart muscle on the one side and the execution of a large amount of work by the heart on the other, which is made conditional by the heavy body of the child, in that it obstructs respiration by deposits of fat in the abdomen and chest, a condition which, with more violent bodily exertion, may give rise to refusal of the heart to continue.

Treatment.—The treatment of cardiac weakness in children differs little from that of adults. Heart tonics are employed on the one hand and on the other physical therapeutic methods, both local and general. As regards the latter, cold may be used in the form of cold coils, ice-bags, heart bottles, a sovereign means for combating acceleration of the heart and pains and at the same time a cardiac tonic of the highest rank. Unfortunately these methods of treatment are not always easily adaptable to small children. Prophylactically, too, children with heart disease should be made accustomed to cold, applied to the heart for a half hour several times a day.

In the treatment of insufficiency of the heart muscle in children, whether the subjective symptoms of dyspnœa and cardiac pain or those of hepatic congestion are most prominent should be considered. In the former case narcotic measures cannot be dispensed with for a time at least, and it should be noted that doses of morphine chosen to suit the age of the child deserve prominence above all other narcotics. Yet morphine should never be given without giving heart tonics at the same time. With a combination of sedative and tonic drugs it is frequently possible to omit the narcotic absolutely for a long time.

Preparations of *digitalis* take the first place among the heart tonics for children. They are indicated in childhood in all conditions of weakness of the cardiac muscle. They first affect the musculature of the left side of the heart, bringing about an invigoration and retardation of the heart contractions, by which the quantity of urine is increased, and the cyanosis and dyspnœa are diminished. The indications for the preparation of digitalis and the length of time it is to be given should be limited very sharply, on account of the cumulative action of digitalis, more to be feared in childhood even than in later periods of life.

Preparations of digitalis are indicated in all conditions of insufficiency of the heart muscle in the course of chronic heart disease and in those acute cardiac affections which are associated with accelerated pulse and diminished blood pressure. The unmethodical administration of digitalis in every heart affection is to be combated. In congenital heart lesions of infancy, if a communication exists between the ventricles, the administration of digitalis may be directly injurious,

because by an increase of pressure in the left ventricle a larger quantity of blood is carried to the right side of the heart and into the pulmonary circulation, already overworked without that. Congenital heart lesions in children should only be treated with digitalis when symptoms of myasthenia, exceedingly small radial pulse or persistent dyspnœa, exist.

In the cardiac affections of children accompanied with myasthenia, one contraindication to the use of digitalis, from its relation to the arterial system, very frequent in later life, is almost totally lacking. Since digitalis also has a constricting effect upon the blood vessels, it should not be given in diseases of the blood vessels. This contraindication to digitalis occurs in childhood only with congenital narrowness of the arterial system and in rare cases of syphilitic disease of the arteries.

Recent pharmacology has furnished two preparations of digitalis which are of especial value in childhood. Golaz' dialyzed digitalis and Cloetta's digalen. Both preparations have an absolutely reliable and constant action and can be added to milk or other liquid nourishment drop by drop. When the stomach is intolerant, these drugs may advantageously be given in small enemata, several times a day. The Cloetta preparation may also be given subcutaneously, besides, but produces slightly painful infiltrations.

In regard to the dosage of the Golaz dialyzed digitalis, it should be noted that one gram of the dialyzed digitalis (25 drops) corresponds to one gram of the digitalis leaves. In children of the first and second years, six to ten drops of this preparation are given daily; in older children a daily dose of 30 to 40 drops must be given for several days, to get the full effect of digitalis in a short time. For chronic treatment with digitalis, two or three drops daily are given to small children and ten to fifteen drops daily to larger children.

The dose of digalen (*digitoxinum solubile Cloetta*) is in childhood 0.1 to 0.3 c.c. of the liquid preparation three or four times a day, either by the mouth, by enema or as a subcutaneous injection. When the last-named mode of administration is employed, a spot covered by skin which moves easily is to be chosen, such as the back or the thigh.

Marfan's recommendation of the macerated infusion of digitalis leaves for children, 20–40 cg. (3–6 gr.) to 60–100 Gm. (2–3½ oz.) water for children under five years, is no longer so valuable since it is easier to use both of the preparations just mentioned.

Besides, digitalis is employed in childhood as powder, infusion and tincture. Powdered digitalis is given in daily doses of 1 dg. ($\frac{1}{6}$ gr.) for each year of life, as powder, decoction or maceration. Tincture of digitalis, a very unreliable preparation (as many drops as the child is years old, given three times a day) is now superfluous, as the Golaz dialyzed digitalis is so much better.

The effect of digitalis preparations, especially of dialyzed digitalis and digalen, is as constant in childhood as in adults. Under exact medical control the administration of digitalis in childhood is just as free from danger as in adults.

Troitzky's opinions upon the dosage of digitalis in childhood deserve mention, as they depend, not upon the body weight, but upon the size of the heart, the blood pressure and the length of the body, with the following rules for dosage as the result:

At the age of one month $\frac{1}{10}$; to the end of the first half year of lactation $\frac{1}{5}$; at the end of the last half of lactation (12 months) $\frac{1}{3}$ of the average adult dose should be ordered.

In the second year four times as much; in the third year six times as much as at the beginning of the second month, *i.e.*, $\frac{4}{10}$, $\frac{6}{10}$ of the adult dose are given. Single doses are the same for the fourth, fifth, tenth, twelfth, thirteenth and fourteenth years, *i.e.*, $\frac{7}{10}$ of the adult dose.

The doses for the eighth and fifteenth years, and also for the sixteenth and seventeenth years are the same, in the former $\frac{8}{10}$, in the latter $\frac{9}{10}$ of the average adult dose.

There are surrogates for digitalis in children, but no true substitute for it. Here belong adonis vernalis, caffeine, strophanthus, convallaria, sparteine and the theobromin preparations. All these drugs are indicated in childhood in the same conditions as in adults. They may serve to support and prolong the action of digitalis without taking its place. In children with chronic heart disease, with symptoms of myasthenia, these drugs are of value to fill in the unavoidable pauses between the cycles of digitalis.

Should digitalis be administered permanently in the chronic heart affections of children, in small doses, or not? The chronic administration of digitalis as employed in later life in recent years does not meet with approval in childhood. It is much more judicious to give larger doses through several days from time to time, to its full action; then the drug is omitted until the effect of the digitalis has disappeared. However it is very good to fill in these pauses by giving other heart stimulants, such as adonis vernalis or caffeine in suitable doses, of which drugs more is to be said soon.

The preparations of caffeine, caffeine sodiobenzoate, sodiosalicylate and citrate, are well borne by the stomach of children even of the tenderest age, but as a rule produce disturbing insomnia in children with heart disease. They do particularly good work in cardiac weakness and tachycardia in the acute infectious diseases. The dose is from 3 to 6 cg. ($\frac{1}{2}$–1 grain) daily for each year of life. The double salts of caffeine above mentioned may also be given subcutaneously, at the same time that digitalis is used internally.

Much recommended to fill in the pauses in the administration of digitalis are Golaz' dialyzed adonis (given in the same doses as dialyzed digitalis) and Golaz' dialyzed convallaria (3 to 6 drops daily for each year of life). Less effective are the strophanthus preparations (given 3 or 4 times a day, one to five drops for each year of life). Biedert also recommends sparteine sulphate, 2 to 5 cg. ($\frac{1}{3}$–$\frac{3}{4}$ grain) several times a day.

The theobromin preparations, theocin, theophyllin, agurin, urocitral, and diuretin, act chiefly as diuretics and assist the treatment with digitalis in children with dropsy. They are given in daily doses of $\frac{1}{4}$ Gm. for each year of life and are best given in enema on account of their bad taste.

Hochsinger advises the following drug treatment in chronic cardiac myasthenia: 1. In cases without dropsy alternate the administration of dialyzed digitalis (five drops for each year of life) for 4 or 5 days with that of dialyzed adonis or convallaria, in doses given above, for 4 or 5 days. For several days now and then give digalen ($\frac{1}{4}$ to $\frac{1}{2}$ c.c. of the finished preparation three times a day) instead of the dialyzed digitalis.

2. In cases with *dropsy,* besides the drugs just mentioned, internally a theobromin preparation, preferably diuretin or agurin, should also be given by enema. If as a result of long continued drug treatment symptoms of gastric or rectal irritation appear, Cloetta's digalen should be used by subcutaneous injection exclusively, which, while it causes slight infiltrations, never produces abscesses when the injection is given aseptically.

Obstinate ascites will sometimes make abdominal paracentesis necessary in children also. As a rule digitalis will act better after puncture than before. Marfan recommends, besides, the administration of fractional doses of calomel [5 cg. ($\frac{3}{4}$ grain) in five doses at half hour intervals once in two weeks] to prevent cirrhotic changes in the congested liver.

Massage may assist the cardiac tonic and drug treatments when there is œdema of the extremities. Gymnastics are injudicious in children with insufficiency of the heart muscle, though massage of the cardiac region sometimes has a favorable effect upon the subjective symptoms. Venesection is of no value in the treatment of the symptoms due to cardiac congestion in children.

8. ACQUIRED DISEASES OF THE BLOOD VESSELS

1. *Aorta.*—Aortic affections are exceptions, on account of the rare occurrence of arteriosclerosis, although in childhood also aortic aneurysms and inflammatory changes of the vessel have been described by Martin, de la Rue and Marfan. Hochsinger has seen aortitis twice in children with congenital syphilis, aged eight and eleven years. Acute

aortitis does not occur in childhood; the chronic form is also rare and almost always depends upon syphilis. Chronic aortitis with spindle-shaped dilatation of the trunk of the vessel has been observed by Zuber and Merget Guillemot, in recurrent articular rheumatism.

Chronic aortitis causes no other symptoms in children than in adults. Retrosternal pains and attacks of dyspnœa are most prominent. Systolic murmurs over the aorta and in the neck, eventually upward projection of the arch of the aorta itself, form the objective symptoms.

Marfan distinguishes a rheumatic and an atheromatous form of aortitis in children. The former is masked by symptoms of stenosis and insufficiency of the aortic ostium, only it often leads to continuation of the inflammatory process upon the inside covering of the trunk of the aorta, to loss of elasticity and dilatation, with the occurrence of asthmatic attacks (Cadet de Gassicourt). The atheromatous form has repeatedly been observed, even in early childhood (Hodgson, in a child of fifteen months, Moutard and Martin in one of two years). In older children such observations become more numerous.

According to Marfan hypertrophy of the left ventricle is absent for a long time with chronic aortitis in children, thus differing from stenosis of the aortic ostium.

Aneurysm of the aorta has even been observed in the fœtus (Phönomenow, Durante). In an inaugural dissertation De la Rue calls from the tenth to the fifteenth year of age the age of predilection for childhood, yet a case has been described in a child of four months and several cases in the fourth and fifth years of life.

According to Lidell, who has grouped 243 fatal cases of aneurysms according to age, seven occurred in children of from 2 to 5 years; one from 5 to 10 years; two from 10 to 15 years; in all ten during childhood.

Jacobi saw an aneurysm of the abdominal aorta in a child and Fruhwald observed rupture of an aneurysm of the innominate artery into the trachea, after tracheotomy, in a girl aged three and a half years.

Etiologically the acute infectious diseases are to be considered, especially syphilis. The seat of aortic aneurysm in childhood is chiefly the arch of the aorta. Another seat of choice is upon the concave surface of the aorta, very close to where the ductus Botalli, *i.e.*, ligamentum arteriosum, branches off.

All the symptoms well known from the pathology of adults are to be considered in the diagnosis. Radioscopy offers most valuable assistance in the diagnosis of this condition even in childhood.

Therapeutically, for aortitis as well as aneurysm of the aorta, the administration of iodine is most important (0.2–0.5 Gm. (3–7 gr.) of sodium iodide daily).

2. *The Peripheral Arteries.*—As a result of the action of specific microörganisms, an acute inflammation, leading to thrombosis and

gangrene, appears sometimes in the course of an infectious disease, in one or more arteries. This has been observed after typhoid fever, scarlet fever, diphtheria, croupous and catarrhal pneumonia. The femoral artery is the favorite seat of inflammatory thrombosis; in a case observed by Hochsinger in a newborn infant with pneumonia the long thoracic artery showed thrombosis.

These inflammatory arterial thromboses in children must be differentiated from arterial emboli which, while less frequent than in adults, occur nevertheless in heart affections, with the production of intracardial thrombi especially. The most frequent emboli are found in the arteries of the brain.

Sclerosis of the peripheral arteries in childhood occurs almost exclusively with syphilis and has been observed by Berghinz in infants aged seven and eighteen months. Seitz describes its appearance as a result of the acute infectious diseases of children, having observed with it accentuation of the second sound at the aortic area and hypertrophy of the left side of the heart. In general though, this affection belongs to later childhood. Aneurysm of the peripheral arteries, especially of the cerebral arteries, also occurs in childhood, depending chiefly upon syphilis (Crisp, Kingston, Lebert, Oppe). Cranwell observed an aneurysm of the axillary artery in a boy of fourteen years.

3. *The Veins.*—Inflammations and thromboses of the veins, due to infecting microörganisms, are not so rare in childhood as inflammations of the arteries. Here belong sinus thromboses and thromboses of the inferior vena cava, the symptoms of which do not differ from those noted in older individuals. In thromboses of the superior vena cava recovery has been observed after a collateral circulation has developed, a rare occurrence in thrombosis of the inferior vena cava.

Phlegmasia alba dolens has repeatedly been described in childhood, in severe chlorosis, pulmonary tuberculosis and other cachectic processes. Recovery has repeatedly been observed in cases of phlegmasia due to chlorosis. Unruh saw thrombosis of the inferior vena cava in a child of one year, caused by an endothelioma of the wall of the vena cava.

Hæmorrhoids also occur in childhood, as is not surprising when the hereditary, family and racial predispositions to this condition are considered. The affection remains latent during childhood as a rule. But sometimes the symptoms are produced as in adults, pains in the sacrum, constipation, tickling sensations in the rectum and hæmorrhage. The hæmorrhoids may become twisted and inflamed also. In children internal hæmorrhoids are more frequent than external. Houzel, among 500 children in whom search for hæmorrhoids was made systematically, found this condition latent four times. There is no cause for surgical interference in children, just as there is no especial treatment for this condition in childhood.

Dilatation of the veins in the form of true varicosities is not observed in childhood except in the veins of the rectum; yet, on the other hand, there are children in whom one or another subcutaneous vein, or the entire district supplied by one vein, appears to be enormously developed. This is most frequently observed between the third and tenth year of life, in children with delicate coloring, blond or red hair. There are usually much dilated veins of the face, somewhat raised, also plainly marked veins upon the anterior chest wall and the upper arms. The veins on the back of the hand, on the contrary, so often prominent in later years of life, are only slightly dilated. Though these dilated veins do occur especially frequently in pale children, chiefly those with tuberculosis, there are also perfectly normal, full-blooded children with this kind of veins which usually disappear totally in later life.

E. Fournier, Jr. considers a dystrophy of the veins characteristic of hereditary syphilis, as shown by ectasia of the veins of the skull. This is not a general dystrophy but only the effect of congestion due to a syphilitic or rachitic hydrocephalus (see also chapter on "Syphilis").

As an exceptionally rare condition should be mentioned varicose dilatation of the cavernous sinus which was observed by Geissler in a child with defect of the interventricular septum.

DISEASES OF THE THYROID GLAND

BY
PROFESSOR F. SIEGERT, OF COLOGNE

TRANSLATED BY
DR. S. W. KELLEY, CLEVELAND, O.

THE thyroid gland has a certain special interest for the pediatrist. It is of great importance in the physical and mental development of the growing child, and is determinative of the normal functions of the vegetative organs as in adults. All abnormalities, resulting from disturbed or suspended secretion of the thyroid, will naturally show more severe effects upon the entire organism the younger the person in whom they develop. In a child, because of the smallness of its upper thorax-aperture and the possibility of strong compression of the trachea and the larger vessels, even changes in size such as acute swelling or quickly growing benign goitre lead more easily to dangerous symptoms.

The increasing knowledge of the etiologic significance of the thyroid gland in delayed bodily and mental development renders it of great importance to the physician. These conditions depending upon insufficient secretion of the thyroid, are surprisingly amenable to organotherapy. The brilliant results of the same in infantile myxidiocy, even in endemic goitre and cretinism, make a general knowledge of diseases of the thyroid desirable.

The thyroid gland is an organ consisting of two lobes joined by a low bridge, and is situated higher in a child than in an adult, extending from the second to third tracheal rings and is often in the shape of a date seed, in the fold formed by the trachea and the œsophagus. Its smooth tight capsule is frequently infiltrated by a heavier deposit of fat which ends imperceptibly in the subcutaneous layer covering the throat. By palpation it is impossible to arrive with any certainty at a decision upon the size or the existence of a normal thyroid, much less upon the proportions of one very much flattened or misplaced far backward. Statements concerning a missing thyroid gland in a normally nourished child based upon examination during life are therefore worthless. Quite frequently the autopsy shows a normal, even large, thyroid, where during life it could not be demonstrated even by a man of experience. On the other hand, small swellings of the isthmus or of the generally greater right lobe occur in marked venous congestion—especially in diphtheria, whoop-

ing-cough, bronchopneumonia of the rachitic,—and by causing overestimation of the correct size during life are apt to mislead. The physiological importance is explained in the introduction to myxœdema.

The disorders of the normal function of the organ to be considered are congestion, acute and chronic inflammation of the thyroid, hyperplasia (goitre). Conditions requiring surgical interference will not be discussed.

CONGESTION OF THE THYROID

Apart from the physiological swelling which occurs regularly during sleep in some children and is often observed in girls at the beginning of puberty, the transient swelling of the thyroid (classified by Demme, Guillaume, Nivel and Laveran as summer-goitre, school-goitre, garrison-goitre, and repeated in all monographs) belongs under this heading. This is sometimes observed in the bloated neck which is occasionally seen after fatiguing exercise in the warm season of the year with the neck encircled by tight clothing. Medical treatment is never necessary as it always disappears in a short time.

INFLAMMATION OF THE THYROID

Acute inflammation of the thyroid as a primary disease is exceedingly rare (Demme, Stamm). Most frequently it occurs secondarily in the course of infectious diseases. Primarily it occurs after trauma, strangulation, child-birth, or without known cause. Swelling of the thyroid, tenderness, and restricted motions of the neck are the symptoms. Application of cold brings quickest reduction to the swelling. Suppuration requires surgical treatment.

Secondary thyroiditis is not infrequent in the course of the infectious diseases. This is especially observed in the course of typhoid fever, scarlet fever, diphtheria, measles, articular rheumatism, malaria and mumps. Pain and swelling occur in the region of the thyroid gland and an abscess results when resolution does not take place. Typhoid bacilli, streptococci, staphylococci, and pneumococci may be found in pure culture. The termination comes in a few days, generally with the disappearance of all the symptoms. Redness of the skin with fever and appearance of fluctuation with formation of abscess are seen in rare cases and still more seldom atrophy of the gland with its results. The *prognosis* is good, even in the very frequent suppuration in typhoid fever. The *treatment* consists in the use of cold by the application of an ice cravat; when suppuration has taken place, warm compresses and subsequently incision.

Chronic inflammation of the thyroid is uncommonly rare and runs its course without clinical symptoms and terminates in atrophy.

Tuberculosis and syphilis never produce an isolated inflammation of the thyroid gland, and need no special discussion.

GOITRE

Partial hyperplasia of the thyroid, which we call goitre, occurs frequently in children. Sporadic cases are relatively rare. The beginning of the goitre, which generally appears at the time of puberty, may be traced back to fœtal life, and in these cases it is a congenital condition. The ages of six hundred and forty-two boys with goitre from Demme's practice were:

37	up to	1 month
59	from	2 to 12 months
35	from	13 to 48 months
83	from	4 to 7 years
94	from	8 to 10 years
150	from	11 to 13 years
184	from	14 to 15 years

This shows a special frequency in the first year of life and from the eleventh year on.

Females are more frequently affected than males by this disease. The kind of goitre shows a difference in adults, in that fibrous goitres are rare, while follicular and cystic goitres, or both mixed, are prevalent, and the colloid goitres are scarcely ever observed.

The goitre infiltrates the thyroid in circumscribed tumors. Corresponding to the relative size of the thyroid lobes, the tumor, if one-sided, is generally found on the right side. But there occur also isolated nodules originating from accessory thyroids or from the middle lobe.

The **symptoms** are the same as in adults. The shortness of the child's neck, often with large development of adipose tissue, many large blood vessels, and softness of the tracheal cartilage, permit evidences of severe derangement more often and more quickly in a child.

Inspiratory stertorous respiration, with shortness of breath easily induced by bodily exertion or excitement, and hoarse rough voice interrupted by whistling inspiration, appear in the larger goitres which surround the whole width of the front of the neck. Smaller unilateral goitres generally produce no symptoms. Severe signs of suffocation, as in retrosternal, fibroid or pediculated goitre of adults, are exceedingly rare. Stormy symptoms threaten in the metastatic thyroiditis occuring in the course of typhoid, infectious diseases, and septic processes, which quickly lead to swelling of the gland with fever and abscess formation. Follicular or cystic goitre is also dangerous if it compresses on both sides, or, being ring shaped, clasps the trachea. Then softening of the tracheal cartilage may occur or squeezing in of the same with secondary bronchitis and bronchopneumonia, also sudden death.

Goitre and cretinism stand in close connection. The endemic appearance of both on the same ground points to this; furthermore, the frequency of goitre in the cretin shows some relationship. On the

other hand, cretinism is primarily due to absence or morbid impairment or alteration of the thyroid secretion.

It is evident, therefore, that the severest endemic cretinism, the so-called sporadic cretinism, and myxidiocy, are accompanied by the absence or total atrophy of the thyroid. But while goitre is almost never missing in the previous history of the cretin, it almost never occurs in that of the myxidiot. While seventy to eighty per cent. of cretins carry goitres, yet countless patients with large goitres live without damaged intelligence, because that part of the thyroid unaffected by the goitre is sufficient for their bodily and mental welfare.

It is therefore to be concluded that goitre leads to myxœdema, myxiodiocy, or cretinism only if all or almost all of the gland tissue has become incapable of function. The largest goitre, if in connection with normal thyroid gland tissue which secretes normally, runs its course without other than local symptoms. On the other hand loss or deficiency of the thyroid with absence of goitre leads to the severest general symptoms.

The **etiology** is the same as with the adult. In goitre localities a predisposing factor exists—most probably with drinking water as the medium,—a factor which does not withstand boiling. In the sporadic cases heredity is the most important factor. In childhood there seems to exist a disposition to hyperplasia of the thyroid (Virchow) which may have some connection with the relative size and vascularity of the thyroid.

The **diagnosis** is simple. From simple congestion or hyperplasia of the thyroid, goitre is distinguished by its affecting only single parts of the gland or by the form and consistence of the tumor. Enlargement of the lymphatic nodes, salivary glands, cysts, and bronchial tumors can be recognized by their location, and are not common in children (Lücke).

The **prognosis** is favorable, not considering those cases in which the trachea rings are laced in or the very rare retrosternal, or fibrous goitres.

The **treatment** is in the majority of cases not operative. It consists in the administration of iodine in very small doses in every instance.

The thyroid preparations are to be considered in connection with myxœdema and myxidiocy. These give, according to von Bruns, an excellent result, like the administration of the thyroid itself, while von Eiselsberg holds the remaining iodine preparations equally effective. Of the latter there are employed in small quantities, potassium iodide, sodium iodide, 0.05–0.2 Gm. (1–3 gr.) daily, syrup of the iodide of iron, iodipin. Externally, locally, potassium iodide ointment is rubbed in, or iodine tincture painted on. Also the use of iodine-bearing spring-water is advisable, and a sojourn by the sea assists the internal treatment.

Internal medication will bring recovery or freedom from all complaint in ninety per cent. of the cases of goitre in children (Kocher),

but requires occasional repetition in case of the not unusual relapse. The surgical treatment does not belong here and is treated of in the surgical text books.

CONGENITAL GOITRE

Congenital goitre requires special discussion. Although rather commonly observed, congenital goitre generally shows itself as a temporary uniform congestion of the whole thyroid gland, and also as an hereditary hyperplasia with increase and enlargement of the follicles with cyst formation.

Fig. 110.

Congenital goitre.

Demme observed hereditary goitre fifty-three times in six hundred and forty-three cases, mostly with spontaneous retrogression and small size, but in one case the weight was 102 Gm. (3 oz.) instead of the normal 2–3 Gm. (30–45 grains) in the newborn. The cases of Béraud and Danyau are often cited of a goitre of 46 Gm. (690 grains) in a fœtus of 5½ months. Hecker's case of 46 Gm. (690 grains) in a newborn, and the cases of Virchow, Mondini, Friedrich. Even marked difficulties in the delivery of the child from goitre are recorded (Hubbauer, Houel).

In congenital goitre, as in goitre of older children and adults, hypertrophy of the thymus is frequently found.

The congenital goitre consists often—contrary to common goitre—in a uniform alteration of the whole gland, which surrounds the entire neck region from the lower jaw to the sternum. This generally decreases spontaneously and seldom becomes a permanent encircling goitre.

In rare instances severe asphyxia at and soon after birth has been reported. Stenosis of the trachea with secondary bronchopneumonia and dyspnœa, and high-grade cyanosis from compression of the large vessels of the neck with sudden death have been observed.

Besides these general causes and a goitre heredity, birth with face presentation, or atypical course of the thyroid vessels (Virchow), have been mentioned as etiologic factors.

The **treatment** is the same as in goitre of older children. Iodine therapy through the mother's milk is recommended by Mossé and Cathala, who had success by giving thyroid tablets to the mother. Iodipin or potassium iodide should also be considered here.

CRETINISM

By cretinism we understand a physical and mental degeneration, which is distinguished by deficient development, by characteristic anomalies of the integuments, and by the hereditary endemic appearances in goitre-regions. It is caused in all cases by the total or partial absence of the thyroid function which is due to absence, atrophy, or to goitrous degeneration of the thyroid gland. With this view endemic goitre would be placed in the same class of diseases as myxœdema, infantile myxidiocy, and infantilism, contrary to the view of Bircher, Bourneville and Pels. This standpoint is the only right one, on the ground of the anatomical situation, the symptomatology and the often very successful thyroid therapy, especially if begun early.

Fig. 111.

Cretin of Thüringen.

The history of cretinism is old. The study of the works up to the sixtieth year of the last century is rendered difficult on account of associating it with different diseases resembling it, such as idiocy, myxœdema, microcephaly, etc. The extensive literature is found in the articles of Baillanger, Bircher, Ewald, Kocher, and St. Leger. These authors first comprehended and treated endemic cretinism as an individual disease.

Cretinism, which is hereditary in a majority of cases, makes its first appearance analogous to myxidiocy, most frequently toward the end of the first year of life, seldom earlier, frequently perhaps in the second or third year of life or still later. The cretinistic expression of face appears early. The goitre next appears and is generally the size of a cherry or walnut. It is more frequently located on the right side, corresponding to the normally larger right lobe of the thyroid. The goitre which is present in seventy to eighty per cent. of all cretins may be totally absent, even with the very severest forms (Wagner). Very soon the flat, abnormally broad, low skull resting on a short, plump body will be noticed. The deficient growth of the short thick extremities is next observed. The premature ossification of the spheno-occipital suture which was originally accepted as typical by Virchow, belongs at all events to the rare exceptions, as in general all synostoses appear late in cretins.

More characteristic than the skull are the frequent saddle-nose, the irregular thick skin of the skull and face heavily wrinkled on the forehead, the thickening of the mucous membrane of the mouth and

tongue, and the hoarse brute-like voice. Balloon-belly and navel-hernia are not infrequent. The paw-like thickening of the hands and fingers is seldom missing.

On the whole, the skin is pale, sallow, and is often covered with pigment dots. The hair is generally bristly, not thick and the beard and pubic hair remain absent for a long time. The nails are seldom changed. The bony skeleton invariably suffers. The normal row-formed proliferation of the cartilage cells of the epiphyses of the growing bones is entirely missing, and also the long-stretched marrow-sprouts adjacent to the cartilage. The periosteal growth remains normal, the epiphyseal centres of ossification appear late and their union with the bone is delayed. The muscles show an indolent reaction, changing from an insignificant to the highest degree. Also the abdominal pressure imperfectly functionates. Constipation is the rule.

The intelligence shows all gradations of reduction, from impaired perception to total imbecility. Therefore the speech is never entirely intact and may be wholly missing or replaced by grunting, roaring, silly laughter. In how far the frequent deaf-mutism of the cretin is a symptom of central trouble, is often difficult to decide. Smell and taste are likewise in most cases slightly developed.

Aside from a few cretins with spiteful, malicious character, the majority have a confiding and harmless disposition. Indifference, mental and bodily, along with the evident dwarfism and imbecile facial expression, dominate the picture of the disease. Sexual backwardness is always present. As before mentioned, the thyroid shows parenchymatous degeneration in about seventy to eighty per cent. of the cases, in other instances there was found an increase in connective tissue (Orel, Virchow, Curling, Langhans, etc.), a small fat cushion (Stilling) in its place, and in isolated instances macroscopic and microscopic changes could not be demonstrated, but this proves nothing in favor of normal function.

Cretinism frequently occurs in the regions of endemic goitre and is hereditary in high grade like the goitre which accompanies it. It is sharply separated from myxidiocy, which until now has only exceptionally (Bourneville, Railton and Smith, Hertoghe and Paterson, Weygandt, Fuchs) been observed in the same family. Kocher is right in his assertion that cretinism appears only in families which have shown goitre or cretinism through generations. After all, we see in endemic cretinism the sign of gradual degeneration lasting through generations in families which have long lived in goitre regions and whose members have been subject to goitres.

The disease is naturally a lasting one according to the pathologic disposition. Without treatment, it will generally remain from the beginning in a more rudimentary or severe form. As a result the children

will develop to all degrees between slight dwarfism with only slight cretinistic characteristics and passable intelligence to the almost motionless vegetating, offensive-looking cretin destitute of every mental emotion, without capacity to attend to his bodily necessities.

Thanks to organotherapy and general hygiene, we are to-day in a position to prevent the severest form of degeneration.

The **diagnosis** rests on locating the sick child as from a cretin- or goitre-family in a goitre region, the physical and mental backwardness, the eventual demonstration of the goitre, the changes of the skin, especially of the face, the absence of typical myxœdema, with, before all, normal soft parts on hands and feet, and the prevalence of the disease in the same family.

Concerning the limits of myxidiocy, see under that heading.

The severity of symptoms depends on the absence of treatment and in part on the intelligence of the parents. More is to be expected from very early and persistent organotherapy. Cure is never to be expected, but an arrest of the progress of the disease and far-reaching improvement in every direction.

The **treatment** of cretinism must be absolutely organotherapeutic and must always be begun as early as possible. Where goitre is in question, examination of the parents needs to be considered, because from Demme's observations, there are children born free from goitre in families in which all earlier born children had shown congenital goitre. A cretin mother or a mother who has already borne a cretin, should be continually under thyroid medication during pregnancy. In every case of cretinism this should immediately take place as soon as the first signs are noticed.

Magnus-Levy, supported by his own splendid results, insists on organotherapy in every case. It will never bring a complete cure as in myxœdema, never so far-reaching an improvement as in myxidiocy, but there will be a gratifying improvement of the lowered mentality, and above all of bodily growth. Whether fresh thyroid or one of the preparations should be employed depends upon the circumstances in the individual case. On account of the rapid decrease of cretinism, thanks to the water supply and therewith a progressively lessening frequency of endemic goitre, we have rarely to deal with the severest forms of cretinism. The early treatment of the lighter cases promises good results.

MYXŒDEMA

Myxœdema is a disease dependent upon deficient or abnormal function of the thyroid gland. This leads to a doughy-like flabby swelling of the skin of the whole body, in previously healthy children, and is accompanied by a retrogression of the mental and bodily activity of the patient.

The name myxœdema originated with Ord, who increased the first four observations of Gull in 1873 by two, and in a systematic manner pointed out (besides the condition of the skin and the psychic alteration, apathy, dementia) the atrophy of the thyroid, the trophic disturbances of the skin, the falling of the hair, the lessened sensibility and the diminished secretion of the glands. Mosler of Giessen, twenty years before, Charcot of Paris fifteen years, and Wernher of Württemburg four years before presented similar cases. In 1892 J. Reverdin of Geneva and Kocher of Berne simultaneously pointed out that myxœdema appears after total extirpation of the thyroid gland. Desault, in 1800, and somewhat later Rush and Cowper described death in animals and man with cramps and apathy, and dementia, respectively, after extirpation of the thyroid.

Schiff of Geneva, in 1859 and then later in 1884, by many experiments on dogs had established the same results of extirpation of the thyroid, which, however, he could prevent by previous transplantation of a thyroid gland into the abdomen of the animal to be operated upon.

Horsley in England, Hofmeister and Fuhr in Germany, and von Eiselsberg in Austria confirmed the investigations of Schiff.

Bardeleben and his pupil Classen contradicted without success the results of the physiologists and surgeons. The Myxœdema Commission which was appointed in England in 1883 brought light to this question.

After Semon in 1883 had first announced that myxœdema and cretinism are different stages of one and the same disease, the English Commission declared, in 1887, that it is the hitherto unknown function of the thyroid to regulate and condition the growth and functions of the organism and that with lack of existence or insufficiency of function of the thyroid, myxœdema or cretinism will appear; also that after extirpation of the gland, cachexia strumipriva will appear.

Horsley undertook some experimental tests and found an accumulation of mucin in the blood to 0.8 per cent.

Vasale established for the first time, in 1891, that not only transplantation but the subcutaneous and intravenous injection of the thyroid juice prevented and cured beginning results of thyroid ablation.

The feeding of the substance and of the extract of the same respectively, which has now become the common property of the medical world, was begun in 1892 in England and Sweden.

Pure myxœdema in the child is a very rare disease. About the best known case is that of Jaunin.

The **symptoms** are as follows: The *skin*, first of the face, then of the neck, the back, the gluteal region, the upper part of the thighs, the hands and feet takes on a doughy, soft consistence, similar to stiffened thin gelatin solution. The belly becomes pendent and the site of an umbilical hernia.

The forehead is irregularly thickened, strongly wrinkled, and by crying it is drawn into uncommonly ugly folds. The palpebral fissures become smaller, through thickening of the eyelids. The nose appears thick and plump, with the nostrils directed forward. The lips become snout-shaped from the thick swelling. The gums and the mucous membrane of the lips appear bloated and thickened. Likewise the skin over the cheeks is thickened and is often the seat of dry scaling, and, like the whole face, is of a pale wax-yellow color.

The skin of the neck has the greatest spongy thickening. The supraclavicular regions like the region in front of the axillæ are the seat of disfiguring deposits of fat. All contours which are formed by the clavicles and ribs are lost.

The skin of the extremities is most markedly thickened on the back of the hands and feet. The thickening of the calf of the leg is notable, but there is no corresponding increase of muscle substance. The abdomen is always markedly pendent, bloated, and in every marked case there is a navel hernia. The hair has a reddish gloss, is generally bristly, and cannot be brushed smooth. It may be quite sparse, especially on the temples, or it may grow luxuriantly. The whole skin is pale, with little flesh color, feels cool and dry and tends to bran-like desquamation. Perspiration is observed only in the rarest instances in older children and then is only scanty. On the other hand the secretion of the sebaceous glands may be normal or even profuse. The electrical resistence of the skin is increased. The nails are occasionally fissured and of bad shape. If myxœdema occurs in a previously healthy child, dentition will be delayed.

The mind gradually becomes affected. Then a certain slowness of speech occurs, and an obtuseness, and indifference in thought and action which were not there before. In severe forms of the disease, for instance, in postoperative myxœdema, the impassiveness soon extends over the entire person and finally a general apathy, with uncleanliness and incontinence.

On the other hand, the intelligence may remain almost normal and the mind intact. In the severest cases, on the contrary, psychoses will also appear, hypochondria, hallucinations and maniacal conditions. These however were observed almost exclusively among adults and have no special pediatric significance.

The motor apparatus participates, with slow muscle reaction, and lowered or normal electrical excitability. A slow awkward gait is the outward sign.

The **diagnosis** is easy after what has been said. At a very early age it might possibly be mistaken for obesity, but the combination of the skin changes with the mental backwardness gives so typical a picture that he who has really known one case will easily recognize all.

The **prognosis** is good in simple myxœdema.

The **treatment** consists alone in the administration of fresh sheep's thyroid or its preparations, and, as far as possible, a vegetable diet.

Concerning the thyroid gland, it is best personally to show the butcher the organ upon a freshly killed animal, after it is skinned, and point out to him its variable size. Then it will be easy to obtain the glands regularly for the patient, especially if the butcher knows the purpose and result, using a pair two or three times a week, after the connective tissue has been removed and the glands scraped fine and mixed with the yolk of an egg and salt.

The result is shown by the rapid disappearance of the myxœdema, the umbilical hernia, the constipation, and in quick recovery of normal mental function and normal growth. There are many preparations of the thyroid on the market which all display the typical efficiency. Concerning the ill-effects of large doses of all of the preparations, Czerny of Breslau has asserted that without exception they are to be considered the ill-effects of the preparation and that they do not appear when giving the fresh sheep thyroid, even in the largest quantity. I myself never saw any ill effects from the use of fresh thyroids such as those mentioned in the treatment of Infantile Myxidiocy.

INFANTILE MYXIDIOCY, IDIOTE MYXŒDEMATEUSE

(Bourneville)

Infantile myxidiocy is a symptom-complex which in the main consists of a general myxœdema with cessation of physical and mental development as it appears in absence of the thyroid gland or the loss of the same or its function, but with apparently intact thyroid.

The first observations, in the seventies, originated in England from Hilton-Fagge, Gull, Fletcher-Beach, and remained unnoticed. Bourneville, a systematic worker, turned the interest of the French physicians toward infantile myxidiocy in 1880, and only in the last decade of the last century did it find recognition by German pediatrists. Not until this century have observations and publications in the German tongue accumulated.

In all these works the opinion prevails and is not contradicted that the disease is conditional, as abundantly proved, upon congenital or acquired absence of the thyroid. Therefore some authors claimed it should simply be called thyreoaplasia (Pineles) or athyreosis (Selter). On the other hand, I was able in 1893 to present to the Gesellschaft für Kinderheilkunde, photographs and preparations of a typical case of congenital myxidiocy with histologically normal thyroid (von Recklinghausen). The experimental investigations of Kaufmann of Basel, personally communicated, also seem to prove the possibility of experimental myxidiocy with intact thyroid.

The disease, as all authors agree, like all thyroid affections, preponderates in girls. In the majority of cases it is congenital. The acquired cases mostly date back to the early years of childhood. How widely it prevails is yet unknown, as the disease itself is yet unknown to many physicians. England, Germany, France, America, Austria, Belgium, Italy and Switzerland, furnish the material of the published cases. Observations accumulate in the hand of the physician who is interested in such matters, even in countries where it is apparently absent. I myself conjecture a parallel in frequency between myxidiocy and severe forms of endemic rachitis.

The cause of infantile myxidiocy, in so far as it appears as a congenital disease, is entirely unknown. Whether it depends from the beginning upon absence of the thyroid or on its disappearance in fœtal life, is still undecided. That the thyroid may remain perfectly normal macroscopically and microscopically was found in one case (Siegert), which reacted wonderfully to the treatment with thyroid. This case shows that congenital myxidiocy and the existence of the thyroid do not exclude each other.

Fig. 112.

Congenital myxidiocy. Girl four and a half years old.

Chronic alcoholism, tuberculosis, syphilis, arthritis, and mental degeneration of the parents, are brought forward as causes by different authors. A sudden fright of the mother during pregnancy, protracted pregnancy, etc., are missing in the etiology of the most typical cases, the cause of which is unknown to us. All processes which may lead to atrophy of the thyroid, infectious diseases, syphilis, suppurations, and the complete removal of a goitre, may be considered as causative factors in acquired myxidiocy.

CONGENITAL MYXIDIOCY

Typical myxœdema shows itself even in the first months most markedly in the face, in the height of the shoulder girdle, and in the shape of the hands and feet. The child occasionally utters a disagree-

able guttural grunt and makes only a few lazy movements. The saddle-nose, and the snout-shaped mouth with protruding, overlarge tongue, are very noticeable. At the end of the first year the absence of teething, the insufficient development of hair on the temples and eyelids, the absolute absence of perspiration, the lack of growth of body and mind, make a striking clinical picture. Like a lazy animal the child lies, motionless, and stupid. In later years the condition remains about the same, the large fontanelle is not closed and no teeth appear, or they are widely separated, coming singly in abnormal succession with pathologic appearances. The belly becomes voluminous, on account of the obstinate constipation. A navel hernia is never absent. Myxidiots always show a pallor of the skin and mucous membranes, to which the reduction of the hæmoglobin does not always correspond. There is often found a relative increase of the red blood cells with a hæmoglobin content of 70 to 100 per cent. and an increase of the lymphocytes. There will always be found a diminution in the nitrogen absorption and gas exchange. Urea and uric acid are diminished, and the chlorides and phosphates appear to be increased. These changes seem to disappear with the organotherapy.

Fig. 113.

Before thyroid treatment. Girl seven and a half years old in March, 1897.

Fig. 114.

The same child June 9, 1897. Formerly was untidy and could understand nothing. Is now tidy, understands everything and can speak a number of words. Assists about the ward. Is happy and contented.

The body temperature is always lowered, and on this account the patient endures cold badly. The pulse and respiration are often slowed. The pineal gland and hypophysis as well as the thymus gland often show a genuine hyperplasia. All the muscles are flaccid, the bones short and thick, lacking all epiphyseal growth and all enchondrial prolifera-

tion, with normal calcination of existing bones; a type held to be the diametrical opposite of rachitis. So it occurs that rachitis, never observed in myxidiocy, is often enough mistaken for it.

There is no aplasia of the bony skeleton. There is present, however, a chondrohypoplasia, which is present to a marked degree in micromelia, the chondrodystrophy of Kaufmann; on the contrary, in rachitis there is a chondrohyperplasia without sufficient

Fig. 115.

The same child February 17, 1898. From December 25 the medication was stopped for experimental purposes. All symptoms including umbilical hernia returned.

Fig. 116.

The same child July 20, 1899, after 28 months of thyroid. The physical development has improved with mental backwardness.

bone formation. Analogous with idiocy the children are untidy and unclean. This condition continues, not taking into account a very slow growth in height and a meagre first dentition.

The large fontanelle was found open in Bromwell's patient who was twenty-two years old, and in Bourneville's twenty-eight year old case. A body length of 75 cm. at sixteen years with weight of 18 kilos was observed by Telfort Smith, 75½ cm. at eighteen years by Herthoge; I myself observed a child of two and one-half years with 50 cm. Photography reveals the characters of the disease better than all description.

The characteristic appearance of the bony skeleton in myxidiocy

is easily and characteristically shown by means of the X-ray. Myxidiocy shows a typical backwardness in epiphyseal bone growth as well as in the appearance of the bone nuclei from the moment of the onset of the disease.

Entirely contrary to rachitis, the epiphyses themselves show no changes. They end with sharp contour, with convex bow near the joint surface with normal ossification. The cylindrical bones, like the phalanges, are short and thick and remain in the infantile condition.

ACQUIRED MYXIDIOCY

Very slowly, without forewarning or striking symptoms, an absolute standstill of the bodily and mental development takes place. The face becomes bloated and expressionless and is almost as motionless as a mask. The whole body and the extremities become myxœdematous, the hair is stiff and bristling, and the motions lingering, slow, restrained. The voice becomes raucous, and unpleasant. The mental activity sinks quickly to a lower grade. The formerly intelligent features become plump and stiff, and soon are bloated and repulsive. The intelligence, however severely reduced, is less affected the older the child at the beginning of the sickness. Most frequently the speech seems to be involved, is used lingeringly under visible difficulty, with quick disappearance of the vocabulary. An extreme slowness of muscular movements occurs in acquired myxidiocy as well as constipation with formation of navel hernia and loss of perspiration.

The **diagnosis** can be made from what has already been written, together with the illustrations. In differential diagnosis the following are to be considered: Simple myxœdema, high-grade obesity, adiposis of rachitic children, mongolism and micromelia. In myxœdema the dwarf growth—bodily and mental—is missing and the high-grade idiocy. The demonstration of the bone changes of rachitis are alone sufficient to exclude myxidiocy; also and more easily demonstrable is the profuse perspiration of the fat rachitics. Mongolism is distinguished without difficulty by the slightly dwarfed growth, the entirely different distention of the soft parts, conditioned on adiposis. The failure of thyroid therapy, the presence of perspiration, normal hair growth, and an uncommonly lively, uneasy, restless manner, are contrary to the entire apathy of the myxidiot. The micromelic, again, outwardly often similar to the myxidiot, shows thickened soft parts only in the arms and legs, normal hair growth on the genitals, which is always missing in the myxidiot; furthermore normal functions and normal intelligence.

The **prognosis**, thanks to organotherapy, is fair. Brilliant results have been attained. Soon after treatment the teeth appear and there is disappearance of all myxœdematous appearances. The patients

become cleanly, approachable, cheerful and soon learn to walk. The mental restoration, however, differs according to the age of the patient, the duration of the illness, and the age at which the illness began.

Paterson treated a boy of nineteen months, who was taken at the eleventh month, with the result that the boy at the age of six and one-fourth years was a normal scholar. A ten-year-old boy (Railton and Smith) who became sick in the second year, and was in his tenth year when treated with thyroid tablets, was after three months' treatment "wonderfully lively, spoke freely, had learned a short poem." I myself treated a Thüringian girl four and one-half years old, who was affected in her first year, and could neither walk nor speak, but after nine weeks appeared bodily and mentally normal (see Figs. 112 and 117). Generally the cases of congenital myxidiocy with normal bodily development remain at the mental level of a three- to five-year-old child.

Fig. 117.

Result of treatment. Same patient as in Fig. 112 after two and one-fourth months on thyroid treatment.

Therapy.—Prompt and persistent organotherapy is of first importance together with a vegetable diet.

I prefer the administration of fresh sheep's thyroid, which can be easily procured from the slaughter house. This is freed from connective tissue, chopped fine, mixed with a part of the yolk of an egg and a little salt, and given on bread. Infants can be easily given finely powdered tablets, such as older children simply chew. These, however, like all older preparations, may lead to intoxication, the so-called "*thyreoidismus,*" nature shows in the form of nausea, fainting, palpitation, trembling, cramps, erythema, migräine, urticaria. Indeed, fatal cases have been observed (Marfan, Immerwohl, Bourneville, Vermehren, Siegert).

But whether the thyroid substance produces poisoning is more than questionable, after the experiments of A. Czerny, who has fed fresh thyroid ad libitum to children without any side effects. Thyreoidismus in infantile myxidiocy behaves much more like the effect of poisons, which are formed by the decay of the thyroid substances in the tablets. If this is not the case the tablets are harmless. A child two and one-half years old, of Becker, cited by Ewald, took ninety 0.3 Gm. (4½ gr.) thyroid tablets, at one time without any thyreoidismus.

Other preparations to be recommended are those mentioned in the Therapy of Myxœdema. The adminstration must continue persistently,

with free intervals of about two days in a week, as according to the gas-changing experiments of Magnus-Levy, a cumulative effect occurs.

INFANTILISM

In recent years Brissaud of Paris and his pupils, have detached a disease picture from the ranks of the lighest cases of myxidiocy which they designate *infantilism*, hypothyreoidism, or forme fruste of myxidiocy.

The clinical features of this disease are a too long continuance in the juvenile state both of the body and the mind with no sexual maturity. Besides a form with rounding fulness of all soft parts, a thick, somewhat bloated face, and a plump body and relatively small extremities (Brissaud type) there is another with slight bone structure, with slender, in fact, over-slender, extremities, narrow, delicate face (Lorain type). Both are based according to Hertoghe, upon insufficient function of the thyroid gland with the hereditary constitutional basis. Apert has published such observations and furnishes the literature up to the present. But all these disease pictures offer to the subjective conviction a wide play for the imagination and are based on uncertain data, as for instance, the absence or atrophy of the thyroid. The most experienced cannot determine positively by palpation the presence of the thyroid in a healthy child in most cases, nor, on the other hand, can he detect the absence of the thyroid in myxidiocy. Therefore it is impossible to give, in the limits of a brief, condensed article, a classification under clinical forms. That very notable results may be obtained in these cases is proved by the authors mentioned; and Hertoghe's illustrations in the Nouvelle Iconographie de la Salpêtrière, are direct proofs.

Fig. 118.

Infantilism. Man, twenty-nine years old.

The radiogram of the hand may be recommended as an absolutely sure diagnosite method. If this shows a backwardness in the appearance of the nuclei of the carpal bones and epiphyses of the metacarpals and phalanges, then the thyroid therapy is always to be recommended. This furnishes a prospect of causing an energetic growth as far as the bony union of the epiphyses and diaphyses are concerned. At the same time the radiogram immediately permits the differentiation of infantilism from rachitic dwarfism, which leads to premature ossification.

BASEDOW'S DISEASE

(Exophthalmic Goitre, Graves' Disease)

Basedow's disease has only limited pediatric interest. It is very rare in childhood, and besides about twenty-five cases in the German literature, there are at the most only about as many more mentioned in the general literature.

The cardinal **symptoms** in a child consist in an enlargement of the thyroid generally greater on the right then on the left side, a pronounced tachycardia and a staring, strange look which usually takes the place of the more rarely pronounced exophthalmos. Frequently there is a systolic blowing murmur over the heart and the great vessels, with strong apex beat and a slight dilatation of the heart. Besides the exophthalmos or glassy, strange, staring look, lagophthalmos sometimes may occur, with infrequent winking (Stellwag's sign), and a retarded intermittent winking of the upper eyelids, which are raised with difficulty in looking upward (Gräfe's sign). The tremor of Basedow's disease as seen in adults is only exceptionally observed, sometimes instead of it there is pronounced chorea. Occasionally there is found intermitting with the vomiting and diarrhœa of the adult, a feeling of great warmth and fever with exceptionally a trifling elevation of temperature. The mind always suffers, although to a much slighter degree than in the adult, and is more marked the older the child.

Besides, a meaningless cheerfulness, irritability, excited disposition, and more seldom a condition of depression, will be observed.

A transition to myxœdema has resulted in a few instances. Generally the patients perspire easily and freely and show according to the moisture an increased electrical irritability (Vigouroux' symptom).

Course.—The course differs from the adult, in so far as an uncommonly rapid development of the disease to the highest point occurs in a few days (Demme, Müller) or weeks, with also a quick disappearance in a few weeks. But in the majority of cases along with the enlargement of the thyroid, are found a very slow high-grade emaciation with the appearance of vitiligo, pigment spots, œdema, and purpura, with longer duration and incomplete recovery or inclination to relapse.

The **etiology** often cannot be fathomed. In isolated cases the goitre of the parents or the patients existed for some time before the onset of Basedow's, or a swelling of the thyroid in the course of an infectious disease (scarlet fever, tonsillitis, whooping-cough). Likewise hereditary influences, cases occurring among brothers and sisters. According to Eichhorst the German race has a predisposition to this disease. Alcoholism of the parents appears with disproportionate frequency in the history. In all cases, however, it deals with an abnormal secretion of the thyroid. The thyroid itself is always enlarged, and, indeed, through di-

rect hyperplasia, a true adenoma growth. Further anatomical lesions, especially of the sympathetic, do not prevail.

Prognosis.—The prognosis is generally more favorable than in the adult. Complete recovery occasionally occurs. In other cases a slight enlargement of the thyroid remains with a tendency to tachycardia, and easy relapse. Deaths without other diseases complicating are rarely observed.

Treatment.—Galvanization is still most frequently practiced, and seems not only to have undisputed suggestive value, but also undeniable results. With a weak current of 2 to 3 M.–A. the anode is applied upon the nape of the neck and the cathode upon the throat at the goitre. Five to ten minutes galvanization is applied at intervals of about two to five days. Of medicaments, weak nervines, bromides and a light vegetable diet. A sojourn in a high climate is beneficial. The most promising internal treatment is to be looked for in the serum of thyroidless sheep, as specified by Möbius, which, prepared by Merck, is to be given in teaspoonful doses in cold milk or a little lemonade. The most promising results have been reported from this rational organotherapy which has in many cases made operative treatment unnecessary.

MONGOLISM

A certain likeness of many idiots to the Mongolian race on account of the slanting inward and downward of the converging palpebral fissures, has led several authors to designate them as mongoloid and compare them with infantile myxidiots. Therefore a brief presentation of the so-called mongolism will be made.

A connection with myxidiocy is possible but it has not been demonstrated. But the patients always, especially at the outset, react in a very remarkable way to thyroid gland therapy.

The Mongols are characterized by an idiotic condition, with a position of the eyes reminding one of the Mongols, generally with open mouth, and often with protruded but not enlarged tongue, thick cushions of fat, especially on the belly, lax joints and flabby musculature and, evident idiocy. Dwarfism does not exist or is scarcely noticed, their manner is always restless, a certain intelligence is present, so that they understand much of the meaning of words and sentences and can utter single words with a rough, indistinct speech. Constipation is seldom absent. Incontinence, least in the waking state, is the rule, and disappears very late.

Teething is sometimes delayed, but complete. Closure of the large fontanelle often takes place in the third to fifth year of life. A saddle-nose appears, but may also be lacking; not seldom a chicken-breast is observed, besides other deformities. H. Neumann describes three cases of valvular heart disease in thirteen mongoloids, Kassowitz two among

seventy-five, and I myself saw one case of pulmonary stenosis in only four patients. Malformations of the external ear, small hernias, high palates are frequent, indeed, cleft palate has been observed. The skeleton generally shows normal proportions, even early ossification; but besides this comes delayed appearance of the bone nuclei and in a striking way chicken-breast, which occurs frequently.

The extreme difference between the skeletons of the mongoloid idiot and the genuine cretin is shown in the following illustrations.

Figure 120 shows the hand of a mongoloid of nine and one-fourth years, only 97 cm. tall at the age of nine and one-half years, in whom the thyroid therapy failed. In spite of the small size, which may have come from the mother, who measured scarcely 1.35 metres, the hand shows itself faultlessly developed. The intelligence of the very restless girl is minimal, constantly talking, although unintelligibly, and can carry out simple orders.

Figure 121, on the contrary, shows the entirely retarded skeleton of the hand of the cretin, illustrated in Fig. 111. The hand at twenty-one years remains in development like that normal in the sixth year of life. Such a retardation always permits the diagnosis of derangement of the thyroid function and generally the prognosis of renewed growth after introducing thyroid therapy.

Fig. 119.

Mongoloid.

The skin of the mongoloids shows nothing excepting slight scaling, which is often observed on the cheeks, and slight chronic eczema on the chin and surroundings of the corners of the lips. Characteristic myxœdematous changes are lacking. Notwithstanding the lively, restless disposition of the idiots, there exists a tendency to obesity, especially of the lower belly and the extremities. The hair and nails show no abnormal appearances. The mucous membranes are normal, and the

great swelling of the mucous membrane of the mouth, etc., in the myxidiot is also missing, like the often disfiguring enlargements of the tongue.

The *intelligence* which is from the beginning only slightly developed, has the tendency to continue on a low level, but still a distinct development is present. The speech is deficient notwithstanding that the mongoloids understand their surroundings very well. They are unclean for many years and never reach the degree of intelligence that appears in the majority of cases of myxidiocy under thyroid gland therapy. A short comparison best shows the great difference between mongoloids and myxidiots. The mongoloid has no symptoms of myxœdema in

Fig. 120.

Mongoloid. Nine and a half years old.

Fig. 121.

Hand of cretin in Fig. 111. Observe the ulna and carpal bones.

later years, no dwarf growth, no apathetic, motionless manner, and none of the cessation in bodily and mental development, like the myxidiot. He is uncommonly lively, soon gets his teeth normally, learns to walk spontaneously, grows symmetrically in a normal way, and never shows the frightful, repulsive appearance of the myxocretin. Toward the thyroid gland medication they behave entirely differently. The mongoloid reacts only in the beginning stage, not in the psychic behavior. Teething, fontanelle closing, obesity, protrusion of the tongue, are immediately and strikingly improved in the myxidiot, only partly, often not at all, in the mongoloid.

The disappearance of the umbilical hernia and constipation after the administration of the thyroid preparations may occur in mongoloids, but in myxidiots it acts much more regularly, and promptly. There is no successful therapy for these idiots. A trial with thyroid leads,

after a beginning improvement, to the disadvantages of this medication. The best line of treatment is intelligent care and education. The idiocy remains permanently.

MICROMELIA

(Chondrodystrophy, Kaufmann; achondroplasia, Parrot; fœtal rachitis of the early authors; micromelia chondromalacia, Marchand)

By micromelia we understand a dwarf growth of the extremities which has no connection with thyroid disease, but depends on congenital disease of the cartilage of the cylindrical bones. The humerus and

Fig. 122.

Micromelia. Girl twenty months old.

Fig. 123.

The same child when two and three quarter years old.

femur remain permanently shortened, while high grade curvatures of all cylindrical bones generally appear but may also be missing.

Myxœdema of the skin, cretinism or idiocy is never observed. Females are especially prone to this disease.

Symptomatology.—The nose is frequently saddle-shaped but the face is intelligent. The compact body with thick fat cushions and un-

commonly short extremities is very striking. The upper arms and thighs are short, the legs bent in the diaphyseal portion outward and forward, besides a high grade of genu valgum or genu varum. The disproportion between the large upper body with big head and the uncommonly small lower body is very noticeable and this is never equalized in the course of growth. The superabundant formation of fat on the lower extremities extends upon the extensor sides, especially from the middle portion of the thighs to the middle of the legs. This forms large folds of adipose tissue, which, however, disappear quickly, as soon as the patients have learned to walk and run. On the early-exercised and often muscular arms there are never so well marked folds of fat. With the exception of symmetrical polydactyly and numerous hip-joint dislocations, few malformations are observed. In no case does the intelligence suffer. The etiology is, as in mongolism, unknown. A successful therapy is entirely wanting.

Index

PAGE

Abscess of liver ... 271
of lung ... 386
tonsillar ... 45
Absorption of carbohydrates ... 281
of fats ... 281
metabolism of ... 279
of nitrogen ... 280
Acetone in recurrent vomiting ... 180
Acidosis ... 286
Acids, poisoning from ... 304
Adenoids ... 50
symptoms ... 51
treatment ... 53
Alcohol, poisoning from ... 306
Amœba coli ... 231
Anchylostomum duodenale ... 238
Aneurism of aorta ... 521
Angina ... 42
catarrhal ... 44
complications ... 46
diagnosis ... 47
etiology ... 42
follicular ... 44
gangrenous ... 49
lacunar ... 44
retronasal ... 45
symtoms ... 43
treatment ... 47
ulcerosa ... 47
etiology ... 48
treatment ... 48
Animal parasites ... 230
Anus, fissure of ... 151
Aorta, acquired diseases of ... 520
congenital narrowness of ... 477
Aortic stenosis, congenital ... 474
symptoms ... 475
Appendicitis ... 213
etiology ... 216
symptoms ... 217
treatment ... 223
Appendicular pain in pneumonia ... 367
Appendix, diseases of ... 213
Arrhythmia ... 455
Arsenic poisoning from ... 305
Arteries, diseases of ... 521
development of ... 436
transposition of ... 478

PAGE

Artery, pulmonary, dilatation of ... 484
Artificial feeding ... 68
cow's milk in ... 71
disturbances from bacteria ... 146
from failure to utilize food ... 140
from insufficient food ... 140
disturbances in ... 69
fat injuries ... 142
gluten injuries in ... 75
milk injuries in ... 73
mortality in ... 69
overfeeding in ... 74, 123
proteid injury ... 142
starch injuries in ... 74, 144
symptoms of overfeeding ... 124
treatment of overfeeding ... 125
Ascaris lumbricoides ... 233
Ascites, chronic tuberculous ... 261
Assimilation, metabolism of ... 283
Asthma ... 353
and bronchitis ... 355
eczema ... 353
diagnosis ... 355
treatment ... 356
Atelectasis of lungs, acquired ... 388
Atresia of anus ... 175
Atrio-ventricular ostia, congenital defects ... 485
Atrophy, infantile ... 85, 135
treatment ... 137

Bacteria, causing digestive diseases ... 77
causing gastro-intestional diseases ... 146
contaminating infant's food ... 119, 146
in cow's milk ... 71, 119
in pleurisy ... 390
intestinal ... 288
normal ... 290
pathological ... 294
staining ... 289
Balantidium coli ... 232
Basedow's disease ... 541
Bednar's aphthæ ... 25
Blood, examinations of, in infants ... 96
in congenital heart disease ... 465
pressure in children ... 413
Bothriocephalus latus ... 243
Bradycardia ... 455

PAGE

Breast-fed infants, colic in 105
digestive disturbances in 65, 97
examination of milk in 92, 113
feeding intervals in 101
inanition in 109
overfeeding in 97
regurgitation in 98
treatment of underfeeding 111
unsuitability of milk in 113
Bronchiectasis 356
Bronchitis 329
diagnosis 333
membranous 332
symptoms 331
treatment 335
Bronchopneumonia 339
course 340
diagnosis 347
physical signs 343
secondary 346
treatment 348

Capillary bronchitis 339
Carbolic acid poisoning 307
Cardiac hypertrophy without lesion ... 456
Catarrh of the large intestine 187
of the small intestine 184
Catarrhal pneumonia 339
Cercomonas intestinalis 232
Cestodes 241
Chloroform, poisoning from 307
Cholera infantum 129
treatment 131
Circulation, diseases of 435
Cirrhosis, atrophic (Lænnec) 272
cardiac 274
cardio-tuberculous 274
from obliteration of bile ducts 275
hypertrophic (Hanot's) 273
Classification of diseases of nutrition .. 80
Clinical diagnosis of diseases of nutrition 92
Colic in breast-fed infants 105
Colicolitis contagiosa 297
Colitis 187
acute 189
stools in 187
treatment 188
mucous 191
Colon, congenital dilatation of 156
treatment 159
irrigation of 104
Congenital stridor 359
Constipation in older children 192
treatment 193
in appendicitis 218
in pyloric stenosis 201
Coryza (see Rhinitis) 317

PAGE

Cough in bronchiectasis 357
in croupous pneumonia 367
Cow's milk in infant feeding 71, 73
Cretinism 529
treatment 531

Dentition 4
difficult 35
late 36
Diarrhœa in older children 184
Digestion, diagnosis of diseases of 92
disturbances, chronic in older children 186
in artificially fed 69
in the breast-fed 65
physiology of in infants 64
metabolism of 279
Ductus Botalli, persistence of 481
diagnosis of 483
Duodenum, ulcer of 151
Dysentery 187
bacteria in 297

Echinococcus 247
Eczema and asthma 353
Emphysema, interstitial 385
of the lungs 384
symptoms 385
Empyema 398
fetid form 400
Heber drainage in 408
pneumococcus form 399
puncture in 407
necessitatis 410
resection of ribs in 409
streptococcus form 400
thoracotomy in 409
treatment of 407
tuberculous form 400
Endocarditis 497
acute 501
diagnosis 503
symptoms 502
treatment 505
chronic 506
diagnosis 509
symptoms 507
treatment 510
malignant 504
pathology of 500
Enteritis, streptococcus 295
Epithelial pearls in gums 3
Epistaxis 326
Expectoration in bronchitis 331
Exploratory puncture in pericarditis .. 493
pleurisy 402
Exophthalmic goitre 541

PAGE

Fat injuries in artificially fed infants 142
Feeding in bronchopneumonia 349
intervals in the breast-fed 101, 108
Fissure of anus 151
Fœtal circulation 437
Foreign bodies in trachea and bronchi 360
in larynx 415
Frenum, diseases of 34
shortness of 118

Gangrene of lungs 387
Gas, poisoning from 304
Gastric indigestion 178
treatment of 179
Gastro-intestinal diseases, bacteria in 146
in early infancy 150
in the artificially fed 129
in the breast-fed 121
of older children 178
Goitre 526
congenital 528
treatment 527
Grave's disease 541

Hæmatoma of nasal septum 320
Hæmorroids 522
Harelip 6
Hay asthma or fever 354
Heart, aortic stenosis of 474
congenital anomalies of 460
causes 461
diagnosis 464
changes in the position of 486
in the size of 487
defects of the interauricular septum 469
of the interventricular septum 467
development of 435
fatty degeneration of 515
hypertrophy and dilation of 456
insufficiency of heart muscle 515
lesions, treatment of 487
murmur 444
nature and frequency of diseases of 449
physiology of 440
pulmonary stenosis of 470
radiography of 451
symptomatology of diseases of the 444
treatment of weakness of 517
Helminthiasis (see Animal parasites) 230
Heredity in digestive diseases 117
Herniæ in children 225
diagnosis 227
symptoms 226
Herpes in croupous pneumonia 365, 370
Hirschsprung's disease 156
Histologic changes in diseases of nutrition 86

PAGE

Hydrotherapy in bronchitis 336
in croupous pneumonia 380

Indigestion in older children 178
Infantile atrophy, pathologic changes in 85
Infantilism 540
Infarction of lungs 386
Inguinal hernia 225
Insufficient feeding in breast-fed infants 107
treatment 111
Interauricular septum, defects of 469
Interstitial pneumonia 382
Intertrigo, treatment of 107
Interventricular septum, defects of 467
murmurs 468
Intestinal bacterial flora, pathologic 294
physiologic 290
Intestinal fermentation in artificially fed infants 133
intussusception 165
stenosis and atresia, acquired 164
anatomy 162
congenital 160
treatment 164
tuberculosis (see Tuberculosis) 194
Intestine, length of in diseases of nutrition 84
Irrigation of colon 104, 188
Invagination, intestinal 165
anatomy 167
diagnosis 172
symptoms 169
treatment 174

Jaundice 267
catarrhal 268
infectious 268

Laryngitis, acute catarrhal 418
chronic catarrhal 420
treatment of 420
Laryngoscopy 411
Larynx, burns of 416
diseases of 413
examination of 414
foreign bodies in 415
œdema of 421
papilloma of 416
perichondritis of 422
stenosis of 416
Lavage of stomach 103
Lead, poisoning from 305
Liver, abscess of 271
acute yellow atrophy of 270
amyloid degeneration of 271
diseases of 267

PAGE

Liver, fatty degeneration of 271
tumors of 277
cirrhosis of (see Cirrhosis) 272
Lungs, diseases of 311
neoplasms, of 412
Lymphadenitis after tonsillitis 46

Macroglossia 33
Malformation about rectum and anus .. 175
congenital 5
Megastomum entericum 232
Mercury, poisoning from 305
Mesenteric lymph-nodes, tuberculosis of 197
Metabolism, pathology of 278
Micromelia 545
Mixed feeding 75, 112
Mongolism 542
Mortality, infant in summer 75
Mouth, care of the 4
diseases of 1
epithelial pearls in 3
mucous membrane of 3
primary affections of 7
secondary affections of 29
Myasthenia cordis 515
treatment 517
Myocarditis 511
symptoms 513
treatment 514
Myxidiocy 534
acquired 538
congenital 535
treatment 539
Myxœdema 531
symptoms 532
treatment 534

Nematodes (see Round worms) 233
Noma (see Stomatitis gangrænosa) 16
Nose, bleeding from 326
congenital anomalies of 316
diseases of 316
foreign bodies in 328
Nursing, act of 2
fat cushion in 2
mechanism of 3
Nutrition, diseases of in infancy 60
bacteriologic causes of 77
classification of 78, 80
clinical diagnosis in 92
etiology 64
from bacterial contamination of food 119
from failure to utilize food 140
from insufficient feeding 107, 140
from overfeeding 97, 123

PAGE

Nutrition, pathology of 81
physiology of 108

Œdema of larynx 421
Œsophagus, atresia and stenosis of ... 59
diseases of 56
stricture of 56
treatment 57
cicatricial 58
treatment 58
Opium, poisoning from 308
Overfeeding in artificially fed infants .. 74
in breast-fed infants 79
treatment 101
Oxyuris vermicularis 235
treatment 237
Ozæna (see Rhinitis, chronic atrophic) .. 322

Palate, cleft of hard 6
Parasitic protozoa 231
Parotitis, primary 39
secondary 39
Pathologic changes in disease of nutrition 81
Pericardial adhesion 493
diagnosis 496
treatment 497
Pericarditis 488
symptoms 489
treatment 492
Peritoneum, tumors of 266
Peritonitis, acute 251
chronic nontuberculous 262
gonococcus 255
in infants 258
penumococcus 252
streptococcus 255
tuberculous 259
chronic 261
Perlèche 28
Pharyngitis (see Retronasal angina) ... 45
Pharynx, diseases of 41
necrosis of 49
Phlegmasia alba dolens 522
Phosphorus, poisoning from 305
Pin's sign in pericarditis 491
Pitre's sign in pleurisy 396
Pleurisy 389
diagnosis 400
exploratory puncture in 406
pathology of 392
purulent (see Empyema) 398
rheumatic 397
serofibrinous form 396
symptoms 392
treatment 404
Pleuritic pain in croupous pneumonia 369

PAGE

Pneumococcus peritonitis ... 252
Pneumonia aspiration ... 346
broncho- ... 339
chronic or interstitial ... 382
secondary ... 346
treatment ... 383
croupous (lobar) ... 361
cerebral, symptoms ... 374
diagnosis ... 375
etiology ... 362
physical signs ... 367
prognosis ... 379
symptoms ... 364
temperature ... 371
treatment ... 380
Poisons, chemical transformation of ... 303
general treatment ... 307
inorganic ... 303
Potassium chlorate, poisoning from ... 305
Premature infants, digestion in ... 115
feeding of ... 116
Proteid injury in infant feeding ... 142
Protozoa ... 231
Ptyalin in saliva ... 1
Pulmonary artery, dilatation of ... 484
hernias ... 412
stenosis ... 470
symptoms ... 474
Pulse, changes in ... 453
rapid ... 454
slow ... 455
Pyloric spasm ... 200
symptoms ... 203
stenosis ... 199
anatomy ... 204
hypertrophic ... 200
symptoms ... 201
pathogenesis ... 206
treatment ... 209

Radiography of the child's heart ... 451
Ranula ... 38
Rauchfuss sign in pleurisy ... 395
Rectum, atresia of ... 175
prolapse of ... 153
treatment ... 155
Recurrent vomiting (see Vomiting) ... 179
Resection of ribs in empyema ... 409
Respiration, chemistry of ... 315
in croupous pneumonia ... 366
in congenital heart disease ... 465
Respiratory tract, anatomy of ... 311
diseases of ... 315
physiology of ... 313
Retropharyngeal abscess ... 55
treatment ... 56
Rheumatism and endocarditis ... 497, 506

PAGE

Rhinitis, acute ... 317
atrophic ... 322
treatment ... 324
chronic ... 321
fibrinous ... 318
purulent ... 318
treatment ... 319
with adenoids ... 34
Riga's disease ... 34
Roger's disease ... 468
Round worms ... 233

Saliva, secretion of ... 1
Salivary glands, diseases of ... 38
Sialoadenitis ... 39
Soor (see Thrush) ... 18
Sore throat (see Angina) ... 42
Spine ... 18
Stenosis of intestine ... 160
of larynx ... 424
of trachea and bronchi ... 358
Stomach contents, examination of in infants ... 95
lavage of ... 103
ulcer of ... 150
Stomatitis ... 7
aphthous ... 8
catarrhal ... 8
gangrænosa ... 16
etiology ... 17
treatment ... 18
gonorrhœa neonatorum ... 27
maculofibrinous ... 8
diagnosis ... 11
etiology ... 10
pathogenesis ... 9
symptoms ... 10
treatment ... 12
ulcerative ... 12
etiology ... 14
symptoms ... 15
treatment ... 15
Stools, examination of infants ... 94
in artificially fed infants ... 127
in breast-fed infants ... 94
in colitis ... 187
constipation ... 193
Streptococcus enteritis ... 295
peritonitis ... 255
Stridor, congenital ... 359
thymicus ... 471
Struma aërea ... 359
Status lymphaticus ... 432
Subcutaneous injection of salt solution ... 130
Subglossitis ... 33
Sudden death and hypertrophied thymus ... 429

	PAGE
Tachycardia	454
Tænia cucumerina	243
echinococcus	247
nana	244
saginata	242
solium	241
Tape-worms	241
treatment	245
Teeth, care of in infancy	4
changes in shape of	36
Hutchinson's	37
rachitic	36
Thoracentesis	405
Thoracotomy in empyema	409
Thrush	18
course	21
etiology	23
localization	20
treatment	23
Thymitis	433
Thymus, absence of	433
anatomy of	426
and sudden death	429
atrophy of	433
diseases of	426
hyperplasia of	427
inflammation of	433
percussion of	428
syphilis of	433
tuberculosis of	433
tumors of	434
Thyroid, congestion of	525
diseases of	524
enlargement of (see Goitre)	526
inflammation of	525
Tongue, anomalies of	31
coated	31
enlarged	33
geographical	31
tie	118
Tonsillar abscess	45
Tonsillitis (see Angina)	42
Tonsils, anatomy and physiology of	41
diseases of	41
hyperplasia of	49
courses	49
treatment	54
tumor of	55
Trachea, foreign bodies in	360
stenosis of	358
Trichina spiralis	239
Trichocephalus dispar	237
Trichomonas intestinalis	232
Tuberculosis, intestinal	194
frequency	195
prophylaxis	198
symytoms	197
Tuberculous peritonitis	259
chronic	261
treatment	265
Ulcera pterygoidea	25
etiology	26
treatment	27
Ulcer of stomach and duodenum	150
Umbilical hernia	225
Underfeeding in breast-fed infants	107
Urine, examination of in infants	96
Vegetable soup in atrophy	138
Veins, development of	436
diseases of	522
murmurs in	447
Vincent's angina	47
Vomiting, in appendicitis	218
in older children	184
in pyloric stenosis	201
recurrent	179
acetone in	180
causes of	181
treatment	183
Weight of breast-fed infants	109
Wet-nurse, diet of	66
in infant feeding	65